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OTOSCLEROSIS *

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The material for this report consists of four temporal bones from three cases of otosclerosis. The first temporal bone described was from a woman, aged 37, whose case was reported in the ARCHIVES OF OTOLARYNGOLOGY for July, 1929. The published report dealt only with the infectious condition. There was extensive infection of the middle ear, surrounding the stapes and round window, with invasion of the pneumatic spaces. Otitis media and mastoiditis of the right side had developed. Later sinus thrombosis, cerebellar abscess and meningitis developed. With these complications the patient died thirty-three days after the onset of the infection. The original hospital record was lost, but so far as could be ascertained there was no history of previous deafness or ear trouble. No history of family deafness was obtained.

Horizontal sections of the right ear, from above down, revealed a focus of pathologic change (fig. 1) which began at the level of the saccule and disappeared just above the level of the start of the cochlea in the vestibule. It was situated between the tendon of the tensor tympani at the processus cochleariformis, the basal coil of the cochlea, the third part of the seventh nerve and stapes, and the vestibule. In the region of the stapes, it extended backward so as almost to touch the region of the anterior joint. It extended to the perilymph space of the vestibule, but was otherwise surrounded by bone, except at the middle levels, where the thickened subepithelial connective tissue of the middle ear was in contact with it laterally and posteriorly. The connective tissue was also seen to extend irregularly into the focus in this region. The focus was small above, became larger, and then smaller again as it disappeared inferiorly. At its widest part, it measured 2 mm. anteroposteriorly and 1 mm. lateromedially.

Just above the level of the focus, the tissue of the bony labyrinth appeared altered from the surrounding bone. Here some of the bone cells, lacunae and canaliculi and the numerous cartilage remnants stained a deep blue (hematoxylin-eosin). It was in this type of tissue that the upper part of the focus came to view. The deep blue or blue-red appearance of the focus made it stand out, even to the naked eye. The blue predominated in places, especially in the anterior portion, while the red

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dominated elsewhere. It also appeared from the various sections that the staining reaction depended to a certain extent on the time of staining; those sections that had been stained longer with eosin showed the red more extensively, while the blue was evidenced when the hematoxylin predominated.

The focus contained numerous prominent marrow-like spaces filled with connective tissue, cells and frequently large blood vessels (fig. 2).

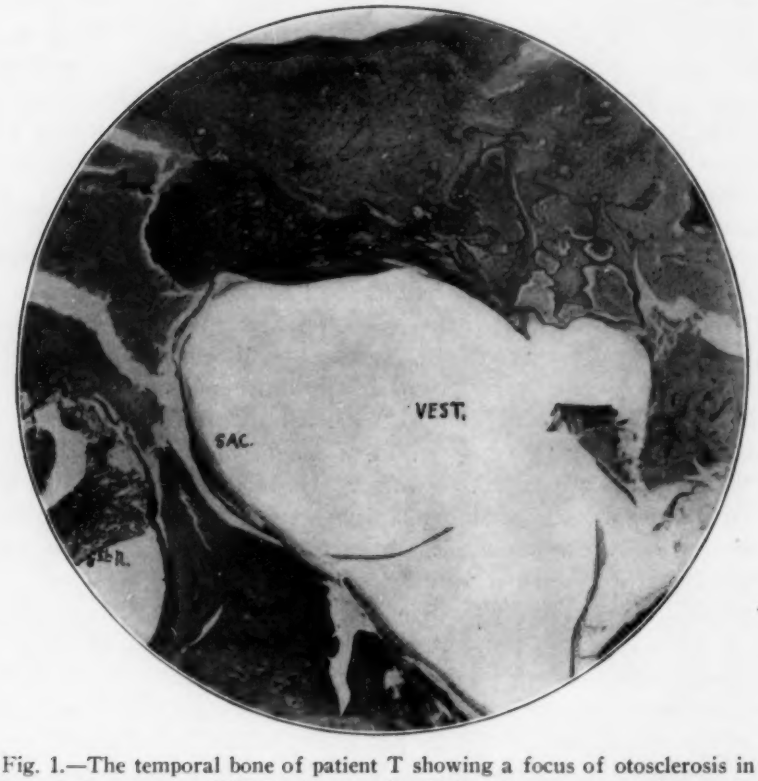

Fig. 1.—The temporal bone of patient T showing a focus of otosclerosis in the region of the stapes. *MM* is the greatly thickened mucous membrane; *S*, the stapes; *F*, the focus of otosclerosis; *Vest.*, the vestibule; *Sac*, the saccule; *8th n*, the eighth nerve; × 12.

Some of the cells resembled fibroblasts, while others, more spherical, could not be definitely identified. In the bluish or more solid parts of the focus, reddish cells could be seen. It appeared that channels had extended from the marrow spaces into the solid part carrying these cells with them. In places in the focus, the bone cells were clumped together in greater numbers than normal. Most of these cells stained a deep blue. The lacunae and canaliculi were well seen in some areas, less well in others. What appeared to be osteoblasts were noted lining the bone

bordering some of the spaces, and these had pale pink-staining tissue just underneath them. The borders of the focus consisted of the marrow-like spaces or the intervening bone. No osteoblasts were noted at these borderlines. In several places, blood vessels connected the focus with the adjacent bone. The vessels were either free or surrounded by several layers of blue-staining bone cells with canaliculi. That portion of the focus bordering on the vestibule seemed slightly swollen into the

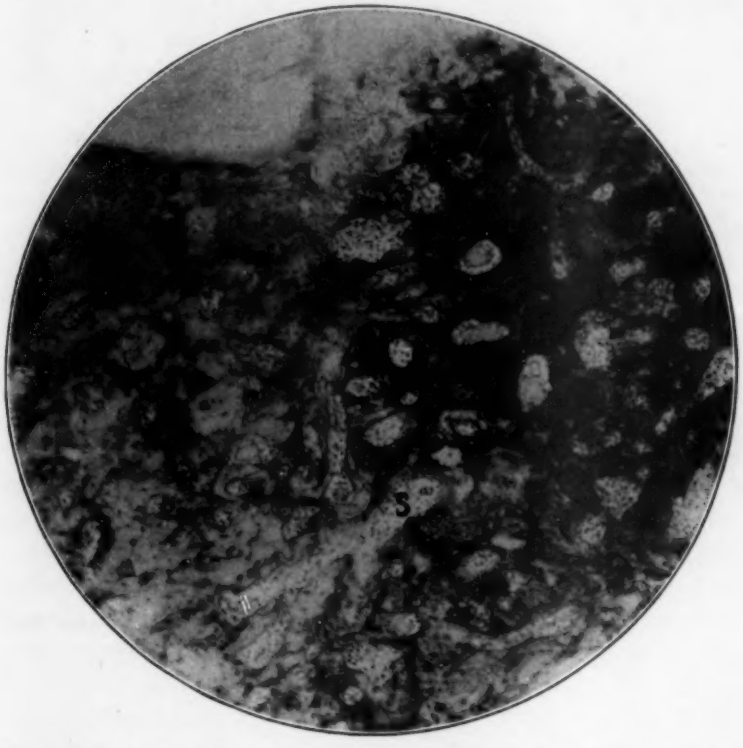

Fig. 2.—A high magnification (× 80) of the focus of otosclerosis in the temporal bone shown in figure 1. *S* represents an enlarged marrow space.

perilymphatic cavity and was lined by endosteum and in part apparently exposed to the perilymph. Of interest was the region between the anterior edge of the focus and the bone surrounding the arch of the internal carotid artery. The bone in this territory showed lamellar formation in only about a dozen places. The bone cells in between these areas were irregularly situated and extremely faint-staining. A considerable number of cartilage remnants were seen in this region, especially close to the focus. Cartilage remnants, however, in addition to being found in the area just mentioned, were found in the modiolus at the basal coil,

all around the cochlea and back of the internal auditory meatus, just medial to the vestibule. The changes in the neuro-epithelium of the cochlea and vestibular organs could not be differentiated from post-mortem changes or those resulting from technic. However, it was evident that there had been no marked, long-standing atrophy.

The temporal bones in the second case were from a young woman, aged 20, patient Fe. She had had slight deafness in the left ear since she was 5 years old. It had followed scarlet fever. Since the onset of the deafness, there had been an intermittent discharge, with a foul odor. There was no history of pain or headache during these years. Ten days before her death, acute otitis media developed in the left ear. Nausea occurred, with headache on the left side and slight vertigo, but no nystagmus. Examination of the hearing was difficult because of the extreme prostration of the patient, but so far as could be determined the left ear was completely deaf, while the hearing in the right ear was questionable. At the hospital, the nausea, vomiting and vertigo subsided for two days, then the headache on the left side began again, and projectile vomiting. At the first operation, a large cavity anterior to the sinus was found, and also a large colesteatomatous mass in the cavum tympani. The radical mastoid operation was performed, and the dura was elevated to the internal auditory meatus. Incision into the cerebellum gave negative results. Following the operation, the condition was fairly good, but there was still slight vomiting, and weakness of the left hand was noted. A second operation was performed, and exploration of the cerebellum again gave negative results. The patient became gradually worse, until death supervened. Autopsy revealed thrombosis of the left lateral sinus and a deep-seated cerebellar abscess (left).

Gross examination of the left or diseased temporal bone showed the absence of most of the external auditory canal and membrana tympani. What was left of this region was partly filled with necrotic material. The mastoid and antrum region had been removed at operation, but the remaining tissue was covered by a necrotic layer. The sigmoid sinus wall appeared infected, and the bulb of the jugular was filled with a thrombus. The right temporal bone seemed normal. The stapes was noted, the drum membrane, malleus and incus having been removed at autopsy.

Microscopic sections of the left temporal bone of patient Fe were made in the horizontal plane from above down and stained with hematoxylin-eosin. The cristae of the semicircular canals, the macula of the utricle and saccule, the organ of Corti, the spiral ganglion and Scarpa's ganglion were in remarkably good condition. This was no doubt due to the early autopsy obtained. The posterior border of the sections showed definite evidence of infection. Here the pneumatic spaces were

filled with organized connective tissue in which were numerous small lymphocytes, some polymorphonuclears and plasma cells. At the extreme posterior edge was a large organized blood clot. Considerable new bone formation with osteoblasts was found lining some of the spaces. The drum membrane, malleus and incus were absent. The skin lining the anterior wall of the external auditory meatus extended posteriorly into the middle ear cavity along its medial wall. Between the skin and the bone of the promontory, etc., forming the medial wall of the cavum tympani, there was considerable connective tissue containing many lymphocytes, some plasma cells, blood vessels and extravasated red blood cells. This was arranged in such a way as to cause pockets to be formed, the pockets being lined by mucous membrane. Anteriorly, goblet cells were seen in the epithelium of the mucous membrane. Most of the stapes had been destroyed, and connective tissue filled in the region of the oval window. The foot-plate was replaced by pathologic bone, which will be described in latter paragraphs. The round window appeared thickened, and on its tympanic side it was surrounded by the overlying organized connective tissue. In the bone adjacent to the anterior wall of the external canal, enlarged marrow spaces were seen, filled with old, dense, fibrous connective tissue. Numerous cartilage remnants were found about the vestibule and cochlea.

One focus of pathologic bone was found in the cochlear region. In the sections cut horizontally from above down, it first appeared just above the cochlea. As the latter was sectioned, the focus extended from the promontory, just lateral to the apical coil, around the cochlea and then at the basal coil turned back to end close to the anterior wall of the internal auditory meatus. Sections lower down showed the portion at the promontory and that at the basal coil persisting to the last, whereas the part which curved around the cochlea had disappeared. The focus stained a blue-red, distinguishing it from the rest of the bone. The edges were peculiar in that they were jagged, thus making the actual limits of the focus more difficult to judge than usual. It was surrounded on all sides by normal-appearing bone, and was separated from the cochlea by a definite layer of healthy bone. The spaces in the focus were not as yet of great size. In some of the spaces, new bone with osteoblasts was seen lining the bony edges. Branching enlarged vessels were seen extending into the normal bone, and these were surrounded by a mantle of several rows of large blue-staining bone cells with canaliculi. Portions of the focus showed extreme disorganization of the bone cell arrangement, but in others haversian-like systems could be made out.

A second focus appeared at the level of the utricle, just lateral to the vestibule and surrounded at its upper levels on all sides by fairly normal-appearing bone. Lower sections showed the focus touching the endos-

teum of the vestibule. The greatest anteroposterior diameter of the focus was 3 mm., and its greatest width lateromedially was 1.5 mm. A band of connective tissue was noted starting at the endosteum, extending in a half curve through the focus and returning again. Lower down, this band broadened out and divided the focus into two parts (fig. 3). The anterior part became a rounded focus anterior to the oval window, while the posterior part became the greatly thickened stapedial foot-plate. The

Fig. 3.—A focus of otosclerosis involving the stapes of the left temporal bone of patient Fe. *S* is the epidermis extending across the medial wall of the cavum tympani, overlying the greatly thickened subepithelial connective tissue, *C.t.; P* shows the normal posterior joint of the stapes; *F* is that part of the focus replacing most of stapedial foot-plate; F^1, that part anterior to the altered annular ligament, *L; Vest.* is the vestibule, and *Sac.*, the saccule; × 17.

cartilage of the stapes in the region of the anterior joint and that of the vestibular border were thus replaced by the pathologic bone, leaving only the thickened annular connective tissue ligament. The back edge of the focus, replacing the foot-plate of the stapes, did not extend into the posterior crus or joint region. The lateral border of the focus was in contact with the vascular connective tissue under the epidermis in the

cavum tympani. Both portions of the focus gradually became smaller as lower sections were cut, so that at the level of the start of the cochlea in the vestibule the focus had disappeared. The focus stained a blue-red, the bone cells and canaliculi taking the blue while the red blood cells, connective tissue, blood vessels and new bone appeared red. The focus contained many large spaces, many more than the focus about the cochlea. The spaces contained connective tissue, blood vessels, cells and osteo-

Fig. 4.—A high magnification (× 312) of the focus involving the stapes in the temporal bone shown in figure 3. *S* is a large marrow space with blood vessels and cells; *OB* is a row of osteoblasts, underneath which the faint staining new bone, *NB*, is seen; *OC* is an osteoclast.

blasts, the latter with pale pink-staining bone underneath (fig. 4). Osteoclasts were also fairly frequently seen. Some haversian-like systems were seen, but otherwise the bone cells were irregularly arranged. In some places, the cells were numerous and distinctly seen; in others, they were scarce and faintly stained. Some blood vessels were seen extending into the normal appearing adjacent bone. The edge of the focus consisted of either the spaces or the intervening pathologic bone between the spaces.

Sections at the level of the crista of the posterior canal were of interest because they showed numerous very tiny, apparently isolated foci. However, perhaps the true condition was that of a single, but branching, focus. The tissue was found in the posteromedial part of the petrous bone, posterior to the crista of the posterior canal and vestibule. The focus seemed to have developed about vascular connective tissue spaces and extended in various directions.

Horizontal sections of the right ear of patient Fe showed the cochlea, utricle, saccule and cristae to be normal, just as on the left side. The spiral ganglion, Scarpa's ganglion and what was present of the seventh nerve were also in good condition. The bone of the petrous portion showed extremely numerous remnants of cartilage. They were present all round the cochlea and between the coils in the modiolus, also in the regions medial and lateral to the vestibule. Normal pneumatic cells were seen posteriorly, and marrow spaces with cells anteriorly.

Of special interest were the foci of pathologic bone found in the sections of the right ear of patient Fe. The sections revealed a large focus anterior to the internal auditory meatus just above the region of the cochlea. Lower sections, at the cochlear level, showed the focus extending from the internal auditory meatus around the basal coil of the cochlea, to the promontory just beyond the apex of the cochlea. This condition persisted to below the cochlear level, where the portions at the promontory and along the side of the cochlea disappeared. However, the basal portion persisted to the lowermost sections. The borders of the focus were irregular. The edge bordering the normal bone consisted either of marrow-like spaces or intervening bone. Vascular connective tissue channels also extended finger-like into the adjacent bone. In some cases, these were surrounded by several rows of blue-staining cells with canaliculi. The focus took the characteristic blue-red stain. The most anterior part was a paler blue, and, having few spaces, had a waxy appearance under the low power lens. Most parts of the focus had enlarged spaces which contained connective tissue, blood vessels, and here and there osteoblasts and osteoclasts. Underneath the osteoblasts, new pale pink-staining bone could be seen. In places, a semblance of haversian systems was seen. For the most part, the bone cells were in patches, numerous or scarce, and very irregularly laid down. A few sections showed the focus in contact with the lumen of the internal auditory meatus and even a small bay formation into the focus at this point.

Medially, but mainly lateral to the medial crus of the horizontal semicircular canal, was an irregular group of small foci, or more likely as found in the left ear, a focus with numerous branchings. The branches seemed to follow vascular channels and stained blue-red with

hematoxylin-eosin. Surrounding the focus was definitely normal-appearing bone. At one point, the pathologic tissue touched the endosteum of the canal. The focus persisted to the level of the posterior crista, where it disappeared.

Just above the level of the stapes, a bridge of normal-appearing bone was seen to extend across the upper part of the fossa of the oval window. At the level of the saccule and just above and anterior to the stapes,

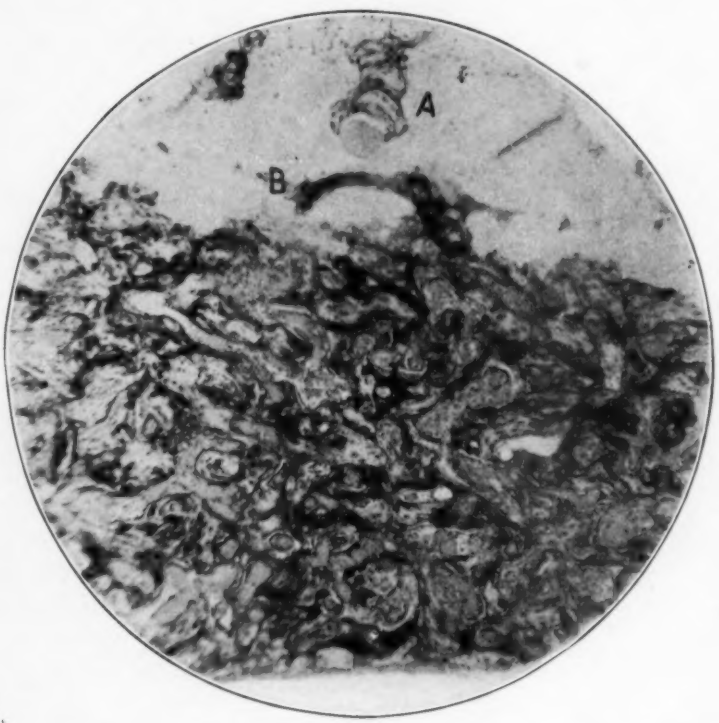

Fig. 5.—A high magnification (× 52) of the focus in the region of the stapes of the right temporal bone of patient Fe. *A* and *B* show the vascular projections which extend out from the focus.

another focus was encountered. At this level the tissue was entirely surrounded by normal bone. The borders were definite, and the edge bordering the normal bone was as described previously for the other foci. In several places, the focus touched cartilaginous remnants. The focus stained a beautiful blue-red and revealed prominent spaces containing connective tissue, large blood vessels, some osteoclasts and osteoblasts with new bone just underneath (fig. 5). The bone cells were scarce and pale-staining in places, while numerous and dark blue in others. Lower levels showed the portion nearest the cavum tympani

to be redder, to contain fewer spaces and to be infiltrated by considerable connective tissue. At the level of the stapes, the focus extended posteriorly just to, but not into the foot-plate. It was separated by a definite layer of normal bone from the cochlea, but extended to the endosteum of the vestibule. The focus increased greatly in its latero-medial diameter, so that there was a distance of 2.5 mm. from the vestibule to the cavum tympani. This lateral growth of the focus appeared to have bent the crura of the stapes downward, but there

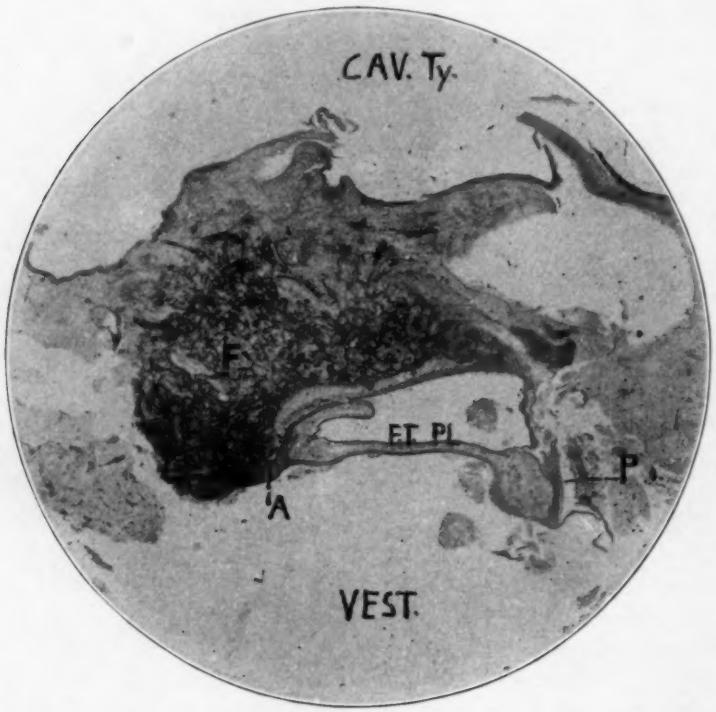

Fig. 6.—The focus in the region of the stapes of the right temporal bone shown in figure 5. *Cav. Ty.* represents the cavum tympani and *Vest.*, the vestibule. The focus, *F.*, extends to the anterior joint region, *A*, but does not invade the stapedial foot-plate, *FT.-PL*, or the posterior joint region, *P.*; × 15.

was no invasion of this ossicle (fig. 6). However, the pathologic tissue had replaced the anterior vestibular lip of the joint. An extension of the connective tissue of the joint was seen, at one level, to extend laterally around the posterior and lateral borders of the focus. Below the level of the stapes, the focus gradually became smaller, the part persisting being anterior and inferior to the oval window. The focus finally disappeared entirely at the level of the round window. At the level at which the spiral ligament takes origin in the vestibule, only the endosteum separated the focus from the spiral ligament.

A final focus appeared at the level of the round window. From a study of the section, the focus appeared to have invaded the bony rim about the secondary tympanic membrane so as to have replaced approximately two thirds of it. The pathologic tissue was in contact with the membrane at the upper and lower levels of the window. The focus took a definite blue-red stain and had fairly definite margins. The spaces were prominent and contained connective tissue, blood vessels, some osteoclasts, and in a few spaces, osteoblasts with new bone formation. The periphery of the focus was as in the other foci described.

In the third case, the temporal bone (left) was from a man, aged 70, patient W. He was found dead in bed, and the coroner's examination revealed organic heart disease, chronic nephritis and senile arteriosclerosis. Seven years before the patient's death, he was examined by Dr. Shambaugh, whose record reads: "Impaired hearing in the right and left ear for forty years, thought to be due to chronic catarrh by the patient. The deafness in the right ear started about the same time as that in the left, and the patient had tinnitus for some years. Paracousis Willisii was present, but not for the last ten to fifteen years. The right seemed to be the best ear, according to the patient. The whispered voice was not heard in either ear, the Weber A was not lateralized. The Schwabach was not prolonged, the Rinne negative on each side. The c4 was much shortened on the right, the galton-whistle at 15 on each side. Diagnosis, otosclerosis."

At autopsy, both the right and the left bones were obtained, but during the preparation the right was so altered that only the left could be studied and described. Grossly, the mastoid, antrum and drum membrane on each side appeared normal.

Horizontal sections of the left ear showed a peculiar tissue in relation to the superior semicircular canal. This disappeared fairly soon, as the bone was cut from above down. The tissue seemed to be mainly connective tissue, no definite bone being demonstrable. Yet the reddish-blue stain with hematoxylin-eosin and the sharply defined margins were suggestive of a pathologic focus. The sections showed the seventh nerve with its geniculate ganglion. The malleus, incus and joint were normal, also the incus-stapes joint. The cochlea and vestibular and organs were noted. As the body had been embalmed several days before the autopsy was made, the neuro-epithelium showed postmortem changes. The drum membrane was intact and normal.

The outstanding feature of the bone was a bony pathologic focus, which was first seen in the sections just above the level of the stapes. It gradually increased in size, and extended into the region of the stapes, so that part of the anterior crus, the joint and most of the footplate were replaced by it (fig. 7). The anterior joint could not be seen, although there was an irregular band of connective tissue simulating

one. What was left of the anterior crus of the stapes was seen attached to the focus. The posterior crus, joint and adjacent part of the foot-plate were uninvolved. As the sections were followed through the level of the saccule to the start of the cochlea in the vestibule, the tumor mass became smaller again. It disappeared at the level of the floor of the vestibule. The focus came close to the cochlea, but did not invade it. In its greatest diameters, it measured 3 by 1.5 mm. It extended

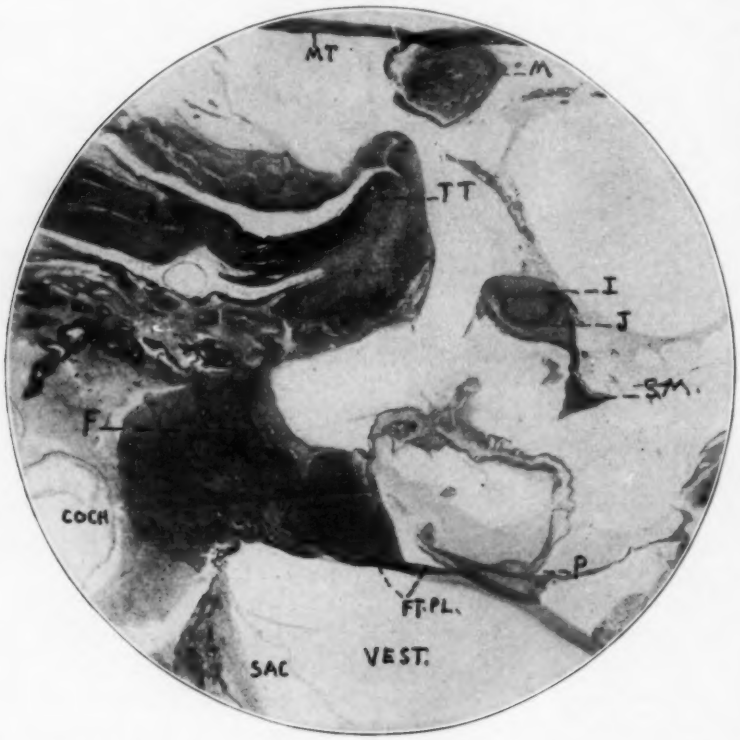

Fig. 7.—A focus of otosclerosis invading the stapes in the temporal bone of patient W. *MT* is the membrana tympani; *M*, the malleus; *TT*, the tendon of the tensor tympani; *I*, the incus; *J*, the malleus-incus joint; *S.M.*, the stapedius muscle; *F*, the focus, which has invaded and replaced part of the stapes foot-plate, *Ft.-Pl.*; *P*, the uninvolved posterior joint; *Coch*, the cochlea; *SAC*, the saccule, and *VEST*, the vestibule; × 17.

laterally to the mucous membrane of the middle ear and inward to the endosteum of the vestibule. The epithelium of the mucous membrane seemed normal, but the underlying connective tissue was thickened and from this a thick band of connective tissue extended into the focus as a false joint. This divided the focus into an anterior and

a posterior part (fig. 8). Between the focus and the cochlea, the bone was faint-staining and contained numerous cartilage remnants. However, this type of bone continued all around the cochlea in a thin layer. Cartilage remnants were also seen in the bone about the vestibule. The pathologic focus took a deep red-blue stain, being less blue and more red than the foci in the other bones. The bone cells stained deep blue in a few places, but were pale in most areas. The cells and canaliculi were everywhere poorly defined, there being prac-

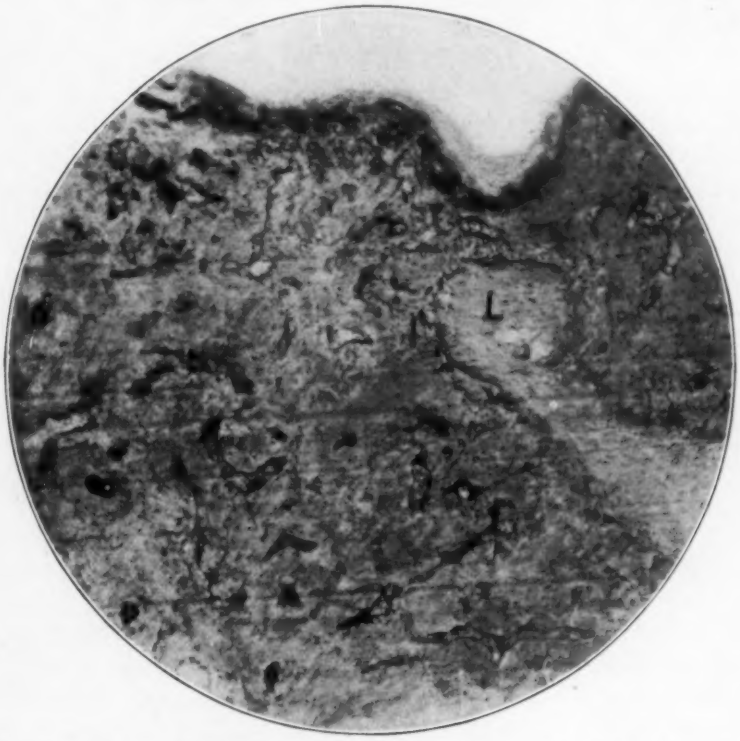

Fig. 8.—A high magnification (× 70) of the focus involving the stapes in the temporal bone shown in figure 7. *L* shows the atypical connective tissue ligament partially dividing the focus.

tically no arrangement into haversian-like systems. A number of vessel-like spaces were seen, which contained either connective tissue and small blood vessels or just connective tissue with only a moderate number of cells. There were no large spaces, therefore, and no osteoblasts or osteoclasts. The borders of the focus were smooth on the tympanic and vestibular sides, more irregular elsewhere, and in places not distinctly outlined from the adjacent normal bone. A few vessels were found extending into the surrounding bone.

SUMMARY

The temporal bone of patient T showed a single focus of otosclerosis in the "site of predilection," but this had not invaded the stapes. The focus contained large spaces and was vascular and cellular. There was little new bone formation or osteoblasts or osteoclasts. An interpretation of the rarefied area just anterior to the focus is difficult. This tissue could have been undergoing chemical changes. Apparently, the case represents a "silent" focus which was producing no symptoms and which was complicated by acute aural infection with fatal intracranial complications.

The left temporal bone of patient Fe showed an extensive focus from the base of the cochlea around to the promontory. A second focus invaded the stapes, while a third was found at the level of the posterior crista, just back of that end-organ. The first two foci, especially, were vascular and contained prominent spaces with a number of osteoblasts, new bone formation and a few osteoclasts. Associated with the foci was evidence of acute and chronic otitis media.

The right temporal bone of patient Fe showed a focus about the cochlea, a second at the oval window but not invading the stapes, a third at the round window and a fourth in the region of the medial crus of the horizontal semicircular canal. Except for the last, the foci showed prominent spaces, were vascular, and contained osteoblasts and a few osteoclasts. The patient therefore had multiple foci in both ears with only a history of chronic otitis media on the left side, with slight deafness.

The left temporal bone of patient W presented a single focus at the "site of predilection." The focus had involved a considerable part of the stapes foot-plate and anterior crus. The pathologic tissue was, on the whole, much more dense than in the other cases, and the bone cells poorly defined. The staining reaction with hematoxylin-eosin was more red and less blue than in the other bones. No osteoblasts, new bone or osteoclasts were noted. This all would conform with the view of a long existing focus. Because of the age of the patient at death (70 years), senile changes may have been present in the hearing mechanism. Unfortunately, autopsy was so delayed as to prevent good fixation of the neuro-epithelium.

COMMENT

Numerous ideas have been put forth as to: (1) the causal factors and (2) the development of the foci. With regard to the causal factors, it is known that the condition frequently occurs in families; also that females are more frequently attacked than males. So far as I know, the youngest child in whom foci have been found was about 1 year of age. It would be interesting to find foci in the ears of

premature infants. This would speak in favor of a hereditary condition as against a postnatal, or acquired, condition. In the embryonic ears that I have sectioned, no evidence of foci has been seen. Some observers have believed that in these cases there is an inherent defect of the ear mechanism or a congenital inferiority of the ear. Such a point has been difficult to demonstrate, because it has been impossible to show changes microscopically to support it. Other observers hold the view that the foci develop from remnants of embryonic tissue. The connective tissue in Cozzolino's zone has been thought to be the starting point of the focus. Yet this does not explain how foci develop in other parts of the petrous bone.

A point of interest concerns the possibility of the foci developing from cartilaginous remnants. Several of the foci in the sections presented appeared to be closely related or actually touching the remnants. However, cartilage remnants were found in regions in which foci are rarely found, if at all, and remnants have been seen in many adult temporal bones showing no foci. Thus the importance of the relationship between remnants and the foci is not easily settled. It has been said that perhaps the foci develop where ossification occurred last. The sections of the ears of embryos, new-born and older infants that I have examined have shown that regions which are frequently cartilaginous when all else is ossified are, in addition to Cozzolino's zone, the regions behind the vestibule in the posterolateral and posteromedial portions of the petrous bone.

Various ideas have been expressed as to what may incite the development of the foci, such as: vasoconstriction of the vessels in the petrous bone, venous stasis of these vessels, chronic local inflammation and chronic local infection. Among several factors influencing the condition may be mentioned: gastro-intestinal toxemia, any focus of infection, syphilis, pregnancy, glands of internal secretion, vascular conditions, tuberculosis, faulty diet (lacking in vitamins) and blue sclera with fragile bones. Those opposed to the view of chronic infection point to the lack of evidence of such infection in the sections, such as the absence of round cells, plasma cells, etc. It may be possible, however, that a previous infection has taken place without leaving permanent changes, the infection having so resolved itself that the microscope is unable to reveal what has gone before.

As to the development of the foci once they are started, there are two main ideas. The first and perhaps the one most frequently mentioned is that there is absorption of normal bone and then replacement by new vascular bone, and that later this becomes denser. The second view is that the focus is a tumor-like formation, and that it destroys the normal bone as it develops. Several points may be discussed con-

cerning the first view. One would expect to find evidence of absorption of the normal bone in the region of the focus. Yet such evidence is definitely lacking in some foci and indistinct in others. It has been said that the changes would be noted if the focus had been sectioned at the proper time in its development. With the limited material at any worker's disposal, it is impossible to answer this question. In the sections that I have seen showing possible changes, the bone adjacent to the focus appeared rarefied. In the absence of osteoclasts, etc., the most likely view is that the alteration was the result of some chemical action. Another matter of note in regard to several of the foci was that there was a crevice-like gap or fracture line between the edge of the focus and the adjacent bone. This was no doubt an artificial gap, and yet it perhaps indicates difference of chemical composition between the bone of the focus and that next to it. The development of the vascular stage was prominently shown in most of the foci, and in the bone of patient W the denser, older stage seemed to be presented.

In the vascular foci, a fair number of osteoclasts and osteoblasts were seen. The latter were situated at the bony edges lining the spaces, and frequently a pink-staining layer of bony tissue was noted just underneath. It seems possible that if this bone building process were to keep up for some years, the tissue would gradually become more dense and the spaces smaller, until finally a dense tissue would result.

The view that the focus is tumor-like has seemed plausible because of the frequent lack of evidence of alteration of bone near the border of the focus. However, the foci do not simulate ordinary tumors in that they do not have a tumor cell common to them. The most characteristic evidence of advance in my sections seemed to be the finger-like vascular projections extending from the focus into the adjacent bone. In some instances these were covered by several rows of blue-staining cells with canaliculi.

Thus, the focus could enlarge by gradually throwing out tentacles into the adjacent bone, and slowly replacing it. The adjacent bone would be prepared, probably by a chemical action. The enlargement of the foot-plate of the stapes indicates that the condition not only replaces previously existing bone but also develops beyond the previous bounds, thus simulating a hyperostosis.

The peculiar staining reactions of the foci interest me. Investigations along this line have developed, so that a separate report will be made concerning these reactions.

In summarizing, it would seem best to state that continued study of otosclerosis should be carried on from all available sources. With a great increase in number of temporal bones sectioned, the missing phases of the condition may be found. The technic of carrying the bone successfully through from start to finish entails considerable time,

patience and experience. For one worker to do all of it himself means that few bones will be examined, while, on the other hand, for technicians to take over much of it entails the danger that valuable leads and observations will be lost.

The material used in this study was supplied by Dr. G. E. Shambaugh, Dr. D. B. Hayden and Dr. J. F. Dickson. Dr. R. Bensley and Dr. G. W. Bartelmez aided in the study of the sections.

DENTIGEROUS CYST OF THE UPPER JAW INVOLVING THE ANTRUM OF HIGHMORE*

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The following case is presented on account of the comparative rarity of this condition, and because there was an opportunity to study in detail the histologic appearance of the material removed at operation.

REPORT OF CASE

Miss D. L., aged 20, consulted me on July 21, 1926, for a swelling of the left cheek, tearing of the left eye and blocking of the left nostril. She was a well developed, well nourished girl, the oldest of a family of eight children, who were all living and well. The family and personal histories were irrelevant.

Since the age of 12 years, the patient had had trouble with her left upper lateral incisor. One year previous to her present complaints, she was treated for about six months for an abscess of the root of this tooth; eventually a pivot tooth was inserted into the root. The patient was wearing this pivot tooth at the time of examination. Seven months before, she was advised by her dentist to consult me because of a slight fulness on the left side of her face. She ignored his suggestion because the swelling was painless and she felt perfectly well. The impulse that prompted her to consult me at last was the fact that her friends were commenting on her deformity, and that she was about to get married.

When she was first observed, there was a marked swelling of the left cheek below the infra-orbital ridge. The nasolabial fold of that side was obliterated, and there was constant epiphora from the left eye.

Rhinoscopic examination revealed that the left inferior turbinate was congested and abutted against the septum. There was a soft fluctuating mass in front of and below the inferior turbinate. The anterior end of the middle turbinate was barely visible. The entire left naris was occluded. The right side of the nose was normal.

Posterior rhinoscopic examination revealed a round, smooth swelling below the inferior turbinate, which occluded the entire left choana. The right choana was normal.

On the left side of the hard palate there was a swelling 2.5 by 1 cm. The surface of this swelling was smooth, covered with normal mucous membrane of the mouth, and fluctuated on pressure; one could palpate a sharp rim of bone all round its circumference. Between the left side of the upper lip and the area just above the left alveolar process, there was another fluctuating swelling, which was continuous with the swelling seen below the infra-orbital ridge. Pressure on the tumor of the cheek transmitted the impulse to the tumor on the hard palate and supra-alveolar region, and vice versa.

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Transillumination of the sinuses revealed a blurring of the left antrum. The light was not completely shut out, but was only faintly transmitted, as compared with the right side.

The roentgen observations were interesting (fig. 1). In the region of the left antrum, there was an oval opacity the size of a large walnut, with a smooth, sharply defined margin. The inner border of this opacity encroached on the left naris, the outer part excavated the zygoma, the upper part shoved up the floor of the left orbit (compare the two orbital cavities shown), and the lower margin hollowed out the vault of the palatal process of the left maxillary bone. In figure 1, the metallic pivot of the left lateral incisor can be seen projecting into this mass.

Fig. 1.—Sinuses of the skull; *P*, pivot tooth, and *C*, cyst

From the foregoing gross observations one had to decide whether the condition was (*a*) a dentigerous cyst, (*b*) osteosarcoma or any other form of malignant tumor or (*c*) empyema of the antrum.

The last-named condition was eliminated at once, because it never causes absorption of bone. Tuberculosis of the upper jaw was excluded, because this condition is characterized by solid granulation tissue.

Osteosarcoma was only provisionally eliminated because of the round, sharply defined shadow of the roentgenogram and the history of prolonged trouble with the root of the left incisor. One could not entirely exclude it, however. At the time of the operation, I had a cartridge of

radium in readiness to insert into the antrum in case the condition proved to be malignant.

The antrum was not punctured, because whatever might be gained diagnostically would be lost either by infecting the cavity or, if a cyst was present, in making the operation more difficult.

Operation.—Under general anesthesia by the intratracheal method, the usual incision for the Caldwell-Luc antrum operation was made. The incision, however was made carefully so as to cut through the mucous membrane only. The mucous membrane was then carefully stripped upward from the underlying fluctuating mass.

A white, patchy capsule presented itself, covering a rounded mass which projected through the anterior bony wall of the antrum and was surrounded by a sharp smooth border of bone.

Figure 2

Figure 3

Fig. 2.—Removed dentigerous cyst. (The scale is in inches.)

Fig. 3.—Longitudinal section of carious tooth with beginning cyst formation at the apex of the root; *a*, cyst wall; *b*, apex of the root; *c*, infection traveling up the root canal and *d*, carious tooth (Oppikofer).

A needle was inserted through the capsule, and 30 cc. of amber-colored fluid, with yellow shimmering crystals floating about in it, was withdrawn. The contents of the second syringe were chocolate-colored.

The tumor was then incised. This revealed a cyst, the inner surface of which was smooth, velvety and brownish. The wall of the cyst shelled out easily from the adjacent bony structures in three large pieces, leaving a large, white, clean bony cavity. The interior of this cavity corroborated the roentgen observations. There was a clear round opening of the anterior wall of the antrum, 2.75 cm. in diameter, which had the appearance of having been made with an instrument. There was a similar deficiency of the hard palate, 7 mm. in diameter. This opening had a shelving edge. The floor of the orbit appeared concave from the interior of the antrum.

The endonasal part of the operation followed. The hypertrophied anterior third of the inferior turbinate was ablated. The inner thin bony wall of the antrum in front was then taken away through the large opening in the facial wall created by the cystic absorption. A large mucoperiosteal flap from below the attachment of the anterior part of the inferior turbinate was dropped on the floor of the antrum. The antrum was lightly packed with iodoform gauze; one end hung out through the outer angle of the labial incision, and the other through the nose. This packing was gradually removed during four days, through the oral end. The patient was discharged from the hospital on the fifth day. There was complete recovery in two weeks. The results were: a normal facial contour, free breathing, no epiphora and a clean nose with a large opening into the antrum below the inferior turbinate.

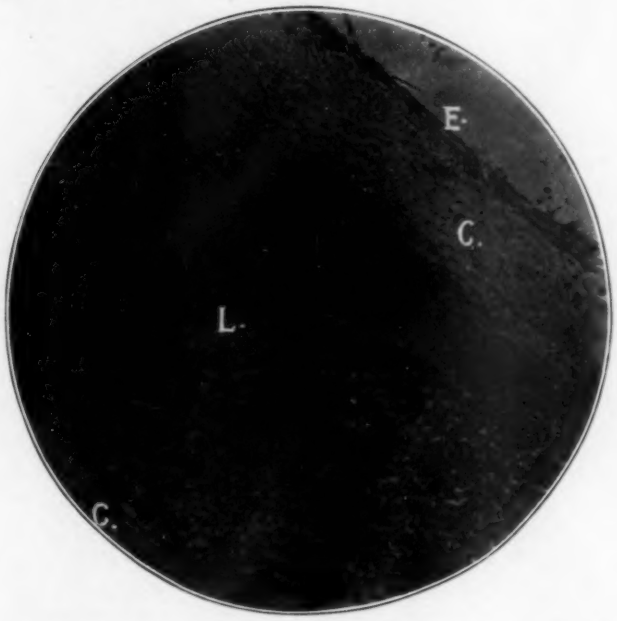

Fig. 4.—Photomicrograph of cyst wall (low power); *E*, epithelial layer of flattened-out squamous cells, *C*, connective tissue and *L*, infiltration of connective-tissue layer with lymphocytes and leukocytes.

Pathologic Observations.—The removed cyst was about 5 by 5 cm. by 2 mm. When reconstructed it was pyramidal, its periphery consisting of thin bone; it was an exact cast of the interior of the antrum (fig. 2).

The outer surface of this cast was mottled; raised white areas alternated with scarred-looking brownish depressions, the whole conveying the impression of the outer surface of a dry bologna sausage. The inner surface was smooth, velvety and of a brownish-chocolate color. The fluid withdrawn from the cyst was sterile. A rich deposit of pure cholesterol crystals was obtained from the sediment.

Histologic Operations.—The cyst wall consisted of an outer layer of bone, a middle zone of connective tissue and a lining of squamous epithelium. This epithelial lining varied in thickness from a few to many cells (figs. 4, 6 and 7).

Fig. 5.—Photomicrograph of cyst wall in the same location as that for figure 4 (high power); *E*, flattened, spindle-shaped epithelial cells invaded by leukocytes, *C*, inner connective tissue layer and *L*, leukocytes, etc.

Fig. 6.—Photomicrograph of cyst wall (low power); *E*, epithelial layer; *C*, inner connective tissue layer; *C2*, outer connective tissue layer; *L*, thick leukocytic layer and *O*, bone.

Fig. 7.—Photomicrograph of cyst wall at different level (low power); *E*, epithelial layer many cells thick as compared with that shown in figures 4, 5 and 6; *L*, extensive infiltration of leukocytes of the connective tissue layer.

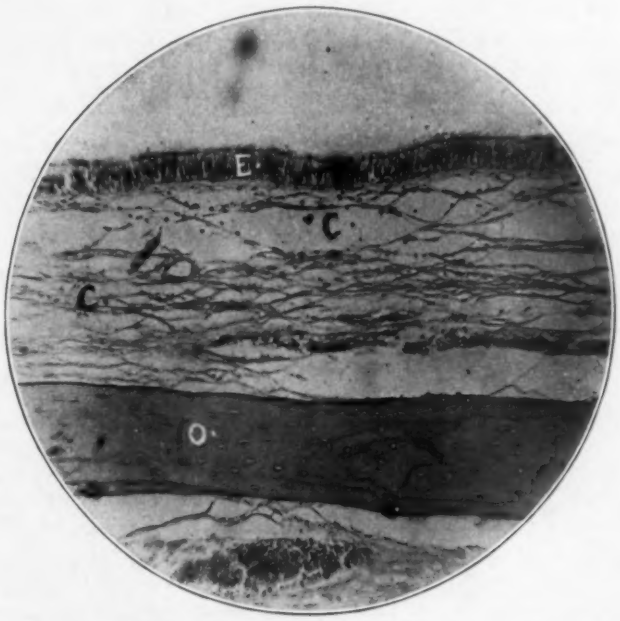

Fig. 8.—Photomicrograph (high power) showing (*E*) ciliated columnar epithelium, (*C*) loose connective tissue and (*O*) bone.

These lining cells were typically squamous or transitional and much flattened out, probably owing to the pressure of fluid in an enclosed cavity (figs. 4 and 5). There was no basal membrane. The epithelial cells rested on a wide band of connective tissue cells, which were closely packed together and arranged in undulating strands (figs. 4 and 6*C*).

This band of connective tissue was divided into two parts by an extensive zone of lymphocytes and leukocytes which outlined a narrow inner band next to the epithelial layer and a wider outer band next to the bony layer (figs. 4 and 6). There were many plasma cells in this cellular zone. An outer thin layer of bone rested on the outer connective tissue band (fig. 6*O*).

This bony structure, at varying levels, showed that the process of bony metaplasia was still going on. There was the characteristic condition of bony apposition and bony resorption with osteoid seams here and there. One of the sections (fig. 8) showed an epithelial layer consisting of ciliated columnar cells, resting on a very loose, areolar connective tissue layer. Next to this layer was bone. This section was from a remaining portion of the antrum wall which had escaped the inroads of the cyst and most probably was from the region of the ethmoid sinus.

It was rather remarkable that a nearly normal antrum wall should be found in the presence of a dentigerous cyst that was of long duration and that involved the antrum extensively.

COMMENT

The dentigerous cyst must be sharply differentiated from the follicular cysts of the maxilla. The latter are embryonic in origin and arise from the so-called Melassez cells, which are nothing more than epithelial cells that are pinched off from the primitive tooth bud. These pinched-off embryonic cell rests from the primitive tooth, lodging in the alveolar process, may get a stimulus toward growth and may develop into a mature follicular cyst which pushes upward into the antral cavity. In this type of antral cyst one usually finds either fully formed teeth or remnants of teeth inside the cystic cavity.

The dentigerous cyst, on the other hand, is the end-result of a cyst of the dental root which in turn is the final stage of a granuloma of the root which is the result of infection carried up the root canal from a carious tooth (fig. 3).

The reason dentigerous cysts are comparatively frequent in the upper jaw and rare in the lower jaw is that morphologically the bone of the upper jaw is less solid than that of the lower jaw, and the presence of the antrum offers no obstruction to their expansion and growth.

The age of incidence of dentigerous cysts ranges from 16 to 54 years. Such cysts are about twice as common in women as in men.

In Oppikofer's series of cases there were fistulas between the alveolar process and the cyst in sixteen instances.

The prognosis for dentigerous cysts is good. When radically removed, they do not recur.

PERFORATION OF THE LARYNX FROM TUBERCULOUS LARYNGITIS

REVIEW OF THE LITERATURE AND REPORT OF A CASE *

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It is generally accepted that laryngeal tuberculosis is almost always secondary to tuberculosis of the lungs. The high incidence of laryngeal tuberculosis as a complication of the disease in the lungs is also well recognized. Jackson and Coates [1] summarized the reports of a number of observers, and they found that the larynx is involved in 12 per cent of the cases of early pulmonary tuberculosis and in 26 per cent of those in which the pulmonary disease is moderately advanced. In the terminal cases, they found that the instances of laryngeal tuberculosis reach 45 per cent or more.

The blood stream, the lymphatic channels and direct implantation from sputum laden with tubercle bacilli are the routes of infection to be considered, and it is still uncertain which of these plays a major rôle, although it is probable that all three are important. The lesion is usually characterized by the formation of tubercles just beneath the epithelium. Several tubercles coalesce, necrosis occurs, and the tubercles break down with the formation of ulcers on the inner surface of the larynx. The true and false vocal cords, the glottis and the aryteno-epiglottic folds are the parts most frequently affected. After ulceration has taken place, secondary infection is common, and aided by other bacteria, the tuberculous lesion may spread to the deeper tissues of the larynx and involve the perichondrium of one or the other laryngeal cartilage. If the cartilage becomes denuded, necrosis results.

With the high frequency of laryngeal tuberculosis and the possible extensive destruction of tissues, it would seem reasonable that in certain instances the process would progress to such an extent that perforation of some part of the larynx would occur and the soft tissues of the neck become involved. When the case about to be reported was first observed,

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* Submitted for publication, April 10, 1930.

* From the Department of Pathology, Yale University School of Medicine.

1. Jackson, C., and Coates, G. M.: The Nose, Throat and Ear and Their Diseases, Philadelphia, W. B. Saunders Company, 1929.

information was sought on the subject in the standard textbooks.[2] Mackenzie [3] mentioned the possibility of abscess formation outside the thyroid cartilage occurring when the cartilage is necrotic from perichondritis. Kyle [4] stated that tuberculous laryngitis may rupture into the pharynx when the cartilages become necrotic. In the various medical indexes, seven cases have been found listed, in which some part of the larynx has been perforated by a process undeniably tuberculous. In this series (group 1) have been placed only those cases which came to necropsy and in which obvious pulmonary tuberculosis was present. In these instances there was no complicating factor which might have been responsible for the laryngeal disease. Tubercle bacilli were not found in any of the cases. Six other instances of laryngeal perforation have been collected (group 2) in which there is a strong probability that the process was tuberculous. In these cases, however, the evidence is not sufficiently conclusive to warrant a positive statement that tuberculosis was the sole cause of the processes observed. One of these cases did not come to necropsy, the patient in another was known to be syphilitic, and in the others either the lungs were uninvolved by any disease or the lesion was not sufficiently typical to be classified as definitely tuberculous. It is interesting to note that between 1836 and 1899 from one to three cases of probable or certain tuberculous ulceration of the larynx with perforation were reported in each decade. Since 1900, no such cases can be found reported in the literature. To review every report of a case of tuberculous ulceration of the larynx on the chance of finding perforation mentioned would be outside the scope of this paper. However, several large series [5] published since 1900 have been read, so that, in all 1,099 cases have been reviewed without finding any reference to perforation of the larynx by a tuberculous process.

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2. Jackson and Coates (footnote 1). Coakley, C. G.: Diseases of the Nose and Throat, ed. 6, Philadelphia, Lea & Febiger, 1922. Phillips, W. C.: Diseases of the Ear, Nose and Throat, ed. 6, Philadelphia, F. A. Davis Company, 1922. Fishberg, Maurice: Pulmonary Tuberculosis, ed. 3, Philadelphia, Lea & Febiger, 1922. McKenzie, D.: Diseases of the Throat, Nose and Ear, ed. 2, St. Louis, C. V. Mosby Company, 1928, vol. 1, p. 131. Kyle, D. B.: Diseases of the Nose and Throat, ed. 4, Philadelphia, W. B. Saunders Company, 1909.

3. Mackenzie (footnote 2, fifth reference).

4. Kyle (footnote 2, sixth reference).

5. Looper, E. A., and Schneider, L. V.: Study of 500 Patients Treated at Maryland State Sanatorium, J. A. M. A. **91**:1012 (Oct. 6) 1928. Hastings, H.: Tuberculosis of the Larynx, Laryngoscope **26**:896 (May) 1916. Fetterolf, G.: The Larynx in Pulmonary Tuberculosis: Analysis of 100 Cases, Laryngoscope **26**:37 (Jan.) 1916. Hayes, E. W.: Laryngeal Tuberculosis; Deductions from Study of 400 Cases in Stonywald and Tucson, Arizona Sanitaria, Southwestern Med. **5**:1 (Aug.) 1921.

CASES REPORTED IN THE LITERATURE, GROUP I

The earliest case found is that reported by Carter [6] in 1848.

A man, aged 35, was first seen two years after the development of persistent cough and night sweats. Eleven months after the cough appeared, an abscess developed in the front of the neck. Three months later, the abscess was opened, but healing was only partial, and one month before death air was respired through the fistula. Three weeks before death there appeared through the thyroid cartilage a second opening which was situated above the first and which allowed air to pass through it freely. Death occurred with all the symptoms of pulmonary tuberculosis. It is stated that hoarseness appeared early in the disease. At necropsy, tubercles were present in both lungs. The laryngeal mucosa was thickened and rough and was detached from the right wing of the thyroid cartilage. The right ventricle of Morgagni was extensively ulcerated, and a second ulcer was present lower down. Both connected with the outside between the halves of the thyroid cartilage, which were completely separated in the midline. Both vocal cords were deeply ulcerated, and the cricothyroid ligament was destroyed.

In 1879, Gaucher [7] gave an account of a case.

A man, aged 63, was the victim of advanced pulmonary tuberculosis and was completely aphonic. Marked dysphagia and a frequent productive cough were present. Cachexia gradually increased. At necropsy, a general miliary tuberculosis of both lungs was found. Many tubercles were present in the larynx, and here ulceration was so extensive that only pieces of the vocal cords were left. The mucosa was entirely gone anteriorly between the vocal cords. The two halves of the thyroid cartilage were necrotic in the midline and were partly separated. The resulting sinus connected with an abscess beneath the muscles on the anterior aspect of the thyroid cartilage.

A full report is given by Dignat [8] in 1883.

A man, aged 24, gave a history of hemoptysis, loss of weight and night sweats. Aphonia was present, and there was a cough which was productive of blood-tinged, purulent sputum. The cough caused great pain in the larynx. Dysphagia was so pronounced that only liquid food could be taken with any comfort. Physical examination showed signs of advanced tuberculosis of the left lung and of the right lung at the apex. Eleven days after the patient's admission to the hospital, there was a sudden accession of laryngeal pain. No food or liquid could be taken by mouth. Aphonia was nearly complete. Death occurred on the seventy-fourth day in the hospital. Necropsy revealed a large tuberculous cavity at the apex of the right lung. Numerous caseous tubercles were present throughout this lung. The upper and middle lobes of the left lung were extensively involved in a tuberculous process. The glosso-epiglottic folds and the posterior surface of the glottis were edematous and contained superficial ulcers. When the esophagus was

6. Carter, A.: Laryngitis-Ulcers Perforating the Thyroid Cartilage, Prov. M. & S. J. London, 1848, p. 298.

7. Gaucher, E.: Tuberculose pharyngo-laryngée necrose et fracture spontanée du cartilage thyroïde, Bull. Soc. anat. de Paris **53**:580, 1878.

8. Dignat, P.: Tuberculose laryngée, necrose et expulsion d'un cartilage aryetinoide, exostose du cricoid ayant formé épine osseuse et perforé l'esophage, Rev. mens. d. laryngol., Paris **4**:239, 1883.

opened, it was found that a perforation was present which formed a communication between the larynx through the left thyroid cartilage. Protruding from the perforation was a piece of bone, which was found to be an exostosis on the superior surface of the cricoid cartilage. The left ventricular band was ulcerated, and beneath it, in the ventricle of Morgagni, was a vast ulceration which laid bare the thyroid cartilage on the left anterior surface and involved the left vocal cord, which had nearly disappeared. This ulcer formed a pus-filled cavity, and a second cavity occupied the site of the left arytenoid cartilage.

Rey [9] in 1885 gave a brief account of a case.

A white man, aged 40, when first seen in July, 1880, was aphonic and had pronounced dysphagia. Emaciation was advanced, and the patient pursued a rapidly downhill course. Death occurred on August 13 of the same year. At necropsy, all stages of tubercle formation were seen in the right lung, and two cavities were present at the apex. A few less advanced tubercles were found in the left lung. The posterior part of the cricoid was necrotic, and here the larynx was in communication with the pharynx. There were small tubercles in the pharynx, surrounding the perforation. Both vocal cords were the seat of small ulcers.

In 1894, Vergniaud [10] reported a case.

A white man, aged 42, had had aphonia and dysphagia for one month before admission to the hospital. On examination, a tumor was found inferior to the hyoid bone. The tumor was not red or hot and resembled a goiter. It was fixed to the thyroid cartilage. On laryngoscopic examination, considerable tenderness was present in the larynx, and the laryngeal mucosa was congested. There were small ulcerations on both vocal cords, and the cords could not be approximated. No deep ulcerations were seen. One month after admission there suddenly occurred great pain in the larynx, and subcutaneous emphysema of the neck and face developed with great rapidity. This subsided in two days, and death occurred two days later. At necropsy, advanced pulmonary and intestinal tuberculosis were found. There was an abscess beneath the thyrohyoid muscle on both sides. The fibrous covering of the thyroid cartilage was stripped up by an accumulation of pus which communicated with the outer abscess and with the larynx at a point between the ventricular folds of the vocal cords in the thyroid angle. There was only slight ulceration of the larynx at this point.

In 1899, Raviart and Marlier [11] presented a case.

A man, aged 23, was admitted to the hospital with signs of far advanced pulmonary tuberculosis. A chronic cough had developed when the patient was 16 years old. Hemoptysis had been observed for six months before admission,

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9. Rey, P.: Cas de phtisie laryngée avec granulations et ulcerations tuberculeuses du pharynx et perforation de la paroi laryngo-pharyngienne, Progrès méd. **1**:355, 1885.

10. Vergniaud: Observation de tuberculose laryngée, carie et perforation du cartilage thyroïde abces tuberculeux extra laryngien; emphysème sous-cutane, Rev. de laryngol. **14**:957, 1894.

11. Raviart et Marlier: Emphysème sous-cutane de la face, cou, etc., du a une perforation du larynx chez un tuberculeux, Bull. Soc. centr. de méd. du Nord, Lille **3**:177, 1899.

and aphonia and dysphagia were present when the patient was first seen. Some time after admission, the patient was found in the morning with marked emphysema of the face and neck, which began following a violent cough. The emphysema spread rapidly to involve the chest and back. Death occurred four days later. Necropsy revealed that tuberculous cavities were present at the apexes of both lungs. Both vocal cords were intact, but running vertically between them was a deep ulcer which had perforated 6 mm. of the thyrohyoid membrane. A quantity of green pus was found in the soft tissues of the neck. There were several smaller ulcerations of the tracheal epithelium. Emphysema of the face, neck, chest and back was noted.

Another instance was reported in 1899 by Schmit.[12]

A white man, 41 years old, died on May 4, 1879. When first seen, he was delirious, and physical examination revealed the presence of extensive pulmonary tuberculosis. There was nothing in the patient's condition to call attention to the laryngeal lesion found at necropsy. Laryngoscopic examination showed only reddening of the epithelium. The voice, though not entirely normal, was typical of ordinary laryngitis, and in his delirium the patient insisted on singing loudly. At necropsy, both lungs were found to be studded with tubercles, and there were large cavitations on the left side. Tuberculous peritonitis and meningitis were present. Outside the thyroid cartilage at its superior edge was a small, circumscribed pocket of pus between the sternothyroid muscles. The abscess communicated with the lumen of the larynx by a fistula between the wings of the thyroid cartilage. On the inner surface, the fistula was a vertical slit which began at the anterior junction of the true vocal cords and extended upward for 2 cm. In the opening was a quantity of pus and a few small sequestrums from the thyroid cartilage. The rest of the laryngeal epithelium was reddened, but not ulcerated. The vocal cords were intact. Externally, the fistula measured 2.5 by 1.5 cm. and reached the upper border of the cartilage.

CASES RECORDED IN THE LITERATURE, GROUP 2

Donné,[13] in 1836, saw a man, aged 50, who at necropsy was found to have an ulcer on the anterior part of the larynx which had perforated the thyroid cartilage. The perforation was accompanied by a phlegmon of the anterior part of the larynx and of the subcutaneous tissue on the anterior surface of the neck. The lungs were apparently normal.

In 1891, McLaren[14] reported the case of a man, aged 41, who had had a productive cough for some time and in whom aphonia and tenderness of the neck had been present for two months. This patient was in extremis when first seen and died shortly afterward. At necropsy, numerous small ulcers were found in the larynx, and the thyroid cartilage

12. Schmit: Laryngite necrosique avec perforation étendue du cartilage thyroïde chez un tuberculeux, Bull. Soc. anat. de Paris **54**:459, 1879.

13. Donné: Ulceration of the Larynx with Perforation of the Thyroid Cartilage, Bull. Soc. anat. de Paris **11**:140, 1836-1837.

14. McLaren, P. J.: Case of Ulceration of the Larynx and Abscess Near the Thyroid Cartilage, New York M. Gaz. **21**:372, 1841.

was perforated anteriorly between the vocal cords. The lungs were the seat of several abscesses. It is stated that the laryngeal ulcers and the abscesses in the lungs were not typical of tuberculosis, but no suggestion as to the probable etiology is made.

Fletcher,[15] in 1851, reported an interesting case.

The patient's age is not given. It is stated that he was known to be syphilitic. Constriction of the larynx with redness and swelling of the neck and aphonia suddenly developed. The respirations were labored. The results of the examination of the chest at this time were unsatisfactory, but the patient's condition improved with the administration of mercury and iodides. The redness of the neck disappeared, and respirations and speech were easier. It was then found that the right side of the chest had the classic signs of tuberculosis. The patient gradually became weaker and died. At necropsy, both lungs were found to be in an advanced state of ulcerative disease. Both vocal cords were destroyed by an ulcerative process. The thyroid cartilage was perforated anteriorly between the vocal cords, the perforation forming a communication between the larynx and an abscess in the soft tissues of the neck. The author believed this to be a syphilitic process, but in the presence of outspoken pulmonary tuberculosis and in the absence of any frank syphilitic lesion, tuberculosis of the larynx seems more probable. The improvement noted with antisyphilitic treatment may well have been due to rest in bed and hospital care.

Thompson,[16] in 1855, described a case.

A woman, aged 51 (in 1852), gave a history of aphonia of some months' duration. When first seen, she had a few small ulcers on the pharyngeal wall. Local treatment brought some relief, and one year later phonation was improved. The next year (1853), she caught cold and had complete loss of voice. Dyspnea became marked, and tracheotomy was resorted to. An abscess developed on the right side of the larynx. This was opened and drained for some time. Shortly afterward it became impossible for the patient to force air through the larynx, though two bits of cartilage and a quantity of pus were coughed up. The area of the abscess began to ulcerate and spread rapidly. Fluid and food were discharged through it. Emaciation became marked, and death occurred in December, 1853. Necropsy revealed that an abscess in the region of the cricoid cartilage had burst near the rima glottidis. One large ulcer had perforated the posterior wall of the larynx and formed a communication with the pharynx. It is stated that the lungs were found to be full of pus, but no mention of tuberculosis is made.

In 1858, Fonsagrives [17] cited a case.

A man, aged 33, had definite pulmonary and laryngeal tuberculosis. A cold abscess developed over the larynx anteriorly. This was incised and drained, after

15. Fletcher: Larynx and Thyroid Cartilage Perforated by Ulceration; the Right Lung in an Advanced State of Ulcerative Disease, Left also Studded, Old Cicatrix at Top of Right Lung, Prov. M. & S. J., London, 1851, p. 577.

16. Thompson, A. H.: Fatal Disease of the Larynx Accompanied by Ulceration and Abscess, Boston M. & S. J. **51**:169, 1855.

17. Fonsagrives: Goitres aeriens survenus brusquement chez un tuberculeux atteint de phtisie laryngée et coincident avec un phlegmon de la partie anterieure du cou, dysphagie et suffocation, disparition spontanée, Union méd., Paris **12**:149, 1858.

which it healed spontaneously. Following this there were intermittent attacks of emphysema of the neck. The air accumulated in the soft tissues of the neck chiefly during coughing and defecation. The emphysema subsided when the patient was at rest. The bouts ceased suddenly, and the patient left the hospital much improved. This is apparently a clear case of tuberculous perforation of the larynx, but in the absence of necropsy observations, no definite statement can be made.

In 1860, Rees [18] reported a case.

A woman, aged 36, caught cold ten months before admission to the hospital. Four months before admission, hoarseness and aphonia developed, accompaned by dysphagia. On admission, the patient presented an ulcer externally over the cricoid cartilage. Tracheotomy was immediately performed, but death occurred eleven days later from lack of nourishment. At necropsy, it was found that the right side of the larynx, from the glottis to the trachea, was nearly destroyed, and the area was occupied by a huge slough. The right ala of the thyroid cartilage was green, necrotic and loose. The right side of the cricoid cartilage was necrotic. Externally, suppuration extended through the thyroid cartilage and into the thyroid gland. The bronchi were filled with purulent material. The description given for the lungs is that they were "inflamed in parts with consolidation here and there." The etiology in this instance is obscure, but the description of the lungs is not incompatible with an acute tuberculous process.

REPORT OF ADDITIONAL CASE

The case described in the following paragraphs was seen on the medical service of the New Haven Hospital.

Clinical History.—E. B., a white ex-sailor, aged 31, was admitted to the hospital on Aug. 23, 1927, complaining of loss of voice and difficulty in breathing. He also stated that he had tuberculosis. Except for influenza in 1918, the past health had been excellent. No childhood diseases or serious illnesses were recalled. No near relatives had tuberculosis. The onset of the present illness was in 1923 with loss of voice, and it was then that the patient was told that he had pulmonary tuberculosis. The aphonia disappeared, but from this time on the patient was confined to his home, except for several sojourns in government sanatoriums. He had spit up blood on numerous occasions. The first large hemoptysis occurred in 1924 and the last one in July, 1927. He had several attacks of aphonia and dysphagia which lasted only a few days. Two days before admission, the patient caught cold, and this was followed by pain in the front of the neck, marked dysphagia, difficulty in breathing and aphonia.

Examination.—On admission, the temperature was 104 F., the respirations were 38, and the pulse rate 160, per minute. The blood pressure was 92 systolic and 60 diastolic. The patient was obviously acutely ill. The respirations were shallow and stridulous, with marked inspiratory difficulty. The face was bathed in perspiration. There was reddening of the skin over the larynx, extending to the right. This region was extremely tender, and to the right of the midline a small, flat, nonfluctuant mass could be felt. The respiratory movements were shallow, rapid and forced, and therefore the lungs were difficult to examine. There was impair-

18. Rees, O.: Strumous Ulceration of the Larynx with Destruction of the Right Half of the Epiglottis, Thyroid and Cricoid Cartilages, Tracheotomy, Death on the 11th Day, Lancet **1**:271, 1860.

ment of percussion resonance at both apexes, extending to the scapular angle on the right posteriorly and to the third rib anteriorly. On the left, the dulness extended to the spine of the scapula posteriorly and to the second interspace anteriorly. The breath sounds over these areas were harsh, with a tubular quality over the right apex posteriorly. Moist râles were heard over the second left interspace anteriorly. The heart action was rapid but regular, and no murmurs were heard. The results of the rest of the physical examination were essentially negative. Later in the day the respirations became more labored, and cyanosis appeared. An examination by means of the x-rays at this time showed well advanced pulmonary tuberculosis throughout both lungs and considerable deviation of the trachea toward the right, beginning at the second dorsal vertebra. On the following day, the temperature was 102 F., and the pulse rate had dropped to 130. Marked inspiratory and expiratory stridor was present, and cyanosis was marked. The apexes of the lungs were flat to percussion, and auscultation revealed that little air was entering the lungs, presumably owing to obstruction of the upper part of the respiratory tract. No râles were heard. Laryngoscopic examination was extremely difficult owing to the poor condition of the patient and lack of cooperation. The impression was, however, that the larynx was essentially normal, and that the obstruction was lower down in the respiratory tract.

Course of Illness.—On August 25, the third day after admission, the temperature dropped to 100 F. in the morning, but at night rose again to 102 F. The pulse remained elevated. On October 26, the temperature fell to 98 F. at noon, but reached 102 F. again at night. The respirations and general condition seemed improved, but the cyanosis was still extreme. On auscultation of the chest, it was found that the patient was depending almost entirely on the right upper lobe for his respiratory exchange. On the fifth day after admission, the temperature was 104 F. in the morning, and the patient was obviously worse. The respirations were extremely labored, and cyanosis had deepened. At 6:30 a. m., Oct. 28, 1927, when the patient was sleeping quietly, he suddenly gasped for breath, became pulseless and was pronounced dead at 6:55 a. m., six days after admission to the hospital and eight days after the acute onset of the present illness. The red blood cell count was 4,650,000 per cubic millimeter; the hemoglobin was 85 per cent; the white blood cell count was 13,200, of which 92 per cent were polymorphonuclear leukocytes; the Wassermann reaction was negative, and the sputum was negative for tubercle bacilli. The clinical diagnosis was: pulmonary tuberculosis, chronic, bilateral, far advanced; lymphadenitis, cervical, chronic, tuberculous.

Pathologic Observations.—The body was well developed and fairly well nourished. The important changes were confined to the thorax and neck. The right pleural cavity was obliterated by firm, fibrous adhesions, and the parietal pleura was firmly adherent to the pericardium. At the level of the second dorsal vertebra, the apex of the lung was firmly adherent to the trachea, pulling the trachea about 3.5 cm. to the right of the midline. The left pleural cavity contained no excess fluid, and the pleural surfaces were smooth and transparent. After the heart was removed, the organs of the neck and both lungs were taken out together (fig. 1). The right lung weighed 550 Gm. It was voluminous and crepitated throughout, except at the apex. The pleura was thickened throughout. There was a large, puckered scar at the apex, and on section in this area the bronchioles were found to be large. Their walls were thickened and had saccular dilatations, giving the appearance of small cavities. The cut surfaces elsewhere presented numerous white tubercles scattered indiscriminately throughout the substance of the lung. The nodules varied from 1 to 5 mm. in diameter and had gray, necrotic

centers. The left lung weighed 400 Gm. There was a puckered scar at the apex, and a hard, conglomerate tubercle could be felt in this region. On section, the nodule was found to measure 1.5 cm. in diameter. It was gray and opaque and had a soft, cheesy center. The lesion was sharply marked off from the rest of the lung by a narrow peripheral ring of gray, translucent tissue. Scattered throughout the lung were numerous small, firm tubercles similar to those in the right lung. On removal of the organs of the neck it was found that the right lobe of the thyroid was enlarged and pushed the larynx and the upper part of the trachea to the left of the midline. When the right lobe of the thyroid was laid back, an abscess cavity was exposed, which was filled with white pus. The abscess was separated from the glandular tissue of the thyroid by a wall of fibrous tissue and from the larynx by the constrictor pharyngeus. On further

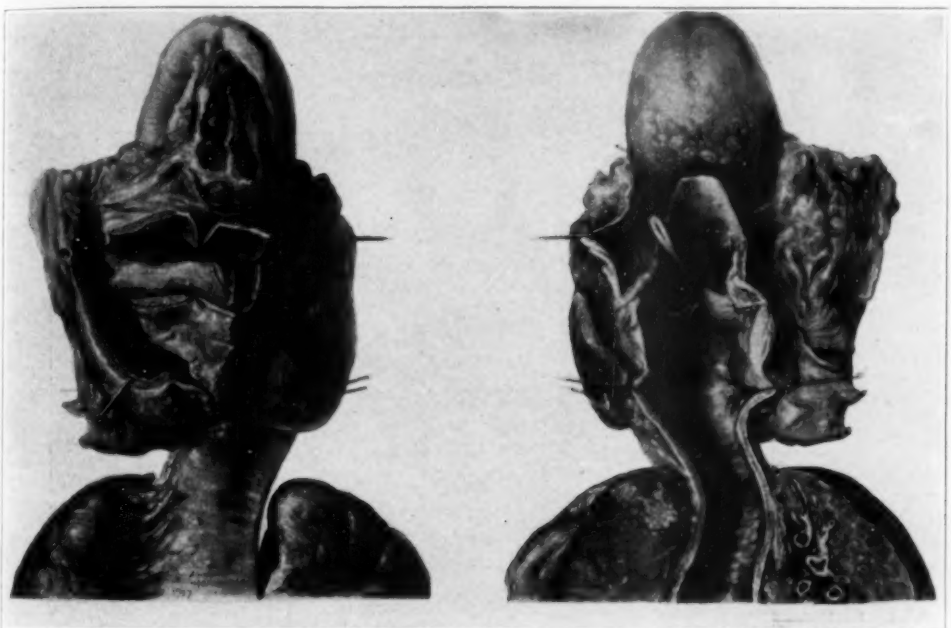

Fig. 1.—Tuberculous ulceration of the left vocal cord with perforation of the thyroid cartilage and abscess formation under the right lobe of the thyroid gland; drawing × ½.

dissection, it was found that the abscess extended up the right side of the neck nearly to the tonsillar fossa. After the organs of the neck were removed, it was seen that the retropharyngeal tissue was extremely edematous and contained a purulent exudate between the swollen strands of connective tissue. The trachea and larger bronchi were filled with fluid pus, and the tracheal epithelium was congested. Before the larynx was opened, it was seen that the vocal cords completely occluded the lumen, so that a small probe could not be passed. After fixation in dilute formaldehyde, the larynx was opened. The left vocal cord was deeply eroded by a large ulcer. The ulcer had heaped-up and undermined edges, and the base had an irregular granular appearance. The pharyngeal epithelium, the aryteno-epiglottic folds and the right vocal cord were greatly swollen. There

was a slitlike perforation in the median line of the anterior laryngeal wall. The perforation extended between the anterior attachments of the true vocal cords just below the thyroid notch. From this point on the external surface of the larynx, there was a sinus tract with fibrous wall which extended beneath the right thyrohyoideus and connected with the abscess beneath the right lobe of the thyroid gland.

Microscopic preparations of the lungs showed varying histologic pictures. At the apex of the right lung only a few alveoli were discernible, and these were larger than is usual. Most of the area was occupied by dense fibrous tissue. The bronchioles in this region were greatly dilated, and most of them contained poly-

Fig. 2.—A section from the edge of the ulcer on the left vocal cord, showing typical proliferative tubercles in the subepithelial tissue; × 100.

morphonuclear leukocytes in their lumen. The bronchiolar epithelium was in varying stages of desquamation. Adjacent to the dilated bronchioles was a deposit of true bone with well formed, marrow-containing canals. In the left apex there was a large circular area, the center of which was composed of homogeneous, amorphous débris with here and there a few shadowy cell outlines. The periphery of the area was composed of small mononuclear leukocytes, fibroblasts and some adult fibrous tissue. Near this area were numerous smaller circular zones which had a similar composition, except that there were frequent multinucleated giant cells at the periphery. Sections from other parts of both lungs demonstrated numerous scattered tubercles composed mostly of relatively dense fibrous tissue, though occasional giant cells were seen, and in a few instances necrosis had taken place. Throughout the sections were focal areas in which the alveoli were packed

with polymorphonuclear leukocytes and fibrin. In the histologic preparations of the left vocal cord, there was considerable subepithelial fibrosis in the edge of the ulcer (fig. 2), and the entire subepithelial tissue was infiltrated with small mononuclear lymphocytes and plasma cells. Just beneath the epithelium were several tubercles composed of fibroblasts, epithelioid cells and lymphocytes. Nearly every tubercle contained one giant cell. The epithelium was entirely gone over the base of the ulcer, and here there was marked fibrosis (fig. 3). Many tubercles were

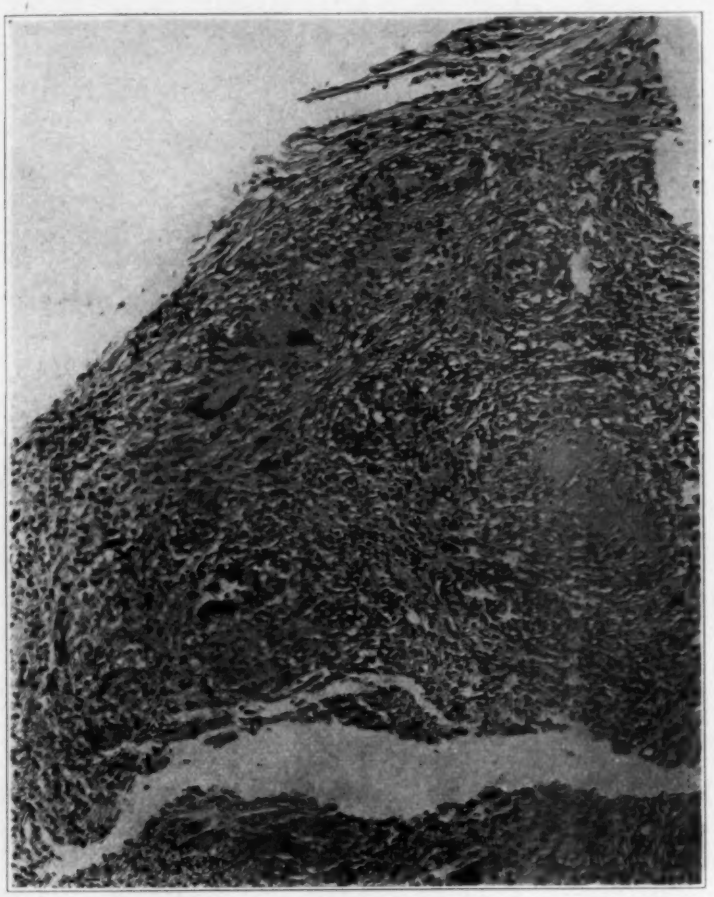

Fig. 3.—A section from the base of the ulcer on the left vocal cord, showing that the covering epithelium is entirely gone. The subepithelial tissue is the seat of an active tuberculous process; × 115.

present in this area. Sections taken through the thyroid cartilage along the line of perforation (fig. 4) demonstrated definite changes in the tissue on both sides of the cartilage. The cartilage itself was eroded. The tissue on the inner surface of the larynx was fibrotic, and many tubercles were present. There was a diffuse lymphocytic infiltration of the tissue, which extended down to the perichondrium of the thyroid cartilage. On the outer surface of the larynx, a similar picture

was encountered, with fibrosis, leukocytic infiltration and tubercle formation. The edge of the sinus tract beneath the thyrohycideus was composed of fresh granulation tissue which was undergoing extensive necrosis. Special stains for tubercle bacilli demonstrated acid-fast organisms in the edge of the ulcer on the left vocal cord and in the tissue anterior to the thyroid cartilage. Culture of the pus beneath the thyroid gland yielded nonhemolytic streptococci and *Staphylococcus aureus.*

Fig. 4.—From the sinus tract extending through the thyroid cartilage, showing erosion of the cartilage. Fibrosis and round cell infiltration of the perichondrium are present; × 90.

Anatomic Diagnosis.—The diagnosis was: chronic proliferative pulmonary tuberculosis (bilateral); chronic bronchiectasis; tuberculous ulceration of the left vocal cord, with perforation of the thyroid cartilage and lateral extension beneath the thyrohyoideus muscle to the soft tissues on the right side of the neck; focal pneumonia; cellulitis of the retropharyngeal tissues; edema of the pharynx, aryteno-epiglottic folds and right vocal cord; occlusion of the larynx.

COMMENT

The condition discussed in this paper is essentially a rare accident which may occur during the course of laryngeal tuberculosis. As has been pointed out, all the reported cases of perforation of the larynx by a probable or certain tuberculous process occurred between 1836 and 1899. The abrupt cessation of such cases since this time might be ascribed to the earlier diagnosis and more successful treatment of pulmonary and laryngeal tuberculosis. The additional case reported from this hospital is that of a man who had a history of laryngeal disease and pulmonary tuberculosis of four years' duration, and who evidently had not had the benefit of a prolonged stay in a sanatorium or even adequate home care. Another conceivable explanation of the sudden cessation of reports in the literature since 1900 is that reports of single cases by pathologists were more popular a generation ago. The condition is one that is more apt to be studied by pathologists than by clinicians, and since it is so rare a happening, no large series could be collected for study.

With respect to age, the instances of perforation in group 1 occurred at about the time that would be expected from a knowledge of the natural history of tuberculosis in general. One person was 23 years old, two were in the fourth decade, four were in the fifth decade and one was 63 years of age. In group 2, the youngest patient was 36 years old and the oldest 51 years. With both groups taken together, all but two of the fourteen instances occurred in men. In the majority of cases (nine in all), the perforation took place anteriorly, between the halves of the thyroid cartilage or through the thyrohyoid membrane. In three cases, perforation was in the posterior part of the larynx, with rupture into the pharynx. Multiple perforations occurred in one case.

It is highly probable that in most cases secondary infection plays an important rôle in the actual perforation. The thyrohyoid membrane represents a weak part of the laryngeal wall from a structural standpoint and it is therefore not surprising that this point should be a frequent site of perforation. Between the halves of the thyroid cartilage in the anterior median line is a second point through which infection might easily spread to the outside of the larynx. With the perforation, there is an extension of the infectious process into the soft tissues of the neck. The changes that take place from here on are variable and may depend largely on whether or not secondary infection is present. If the extension through the larynx takes place slowly and the secondary infection is minimal, the proliferation of fibrous tissue will result in a localized "cold" abscess over the anterior part of the larynx. If perforation is rapid and accompanied by massive secondary infection, the process in all probability not only will involve the tissue anterior to the larynx but may extend

laterally with involvement of the thyroid gland in a phlegmonous process and may even reach the retropharyngeal tissues. It is in this type of case that the development of subcutaneous emphysema must be expected, since the proliferation of fibrous tissue has not been able to keep pace with the extension of the process. In one instance, an abscess over the larynx broke through the skin and discharged on the surface of the neck.

From a clinical standpoint, if an adult with advanced laryngeal tuberculosis has over the larynx a swelling, which may be red and tender or which may be a "cold" abscess, tuberculous perforation of the larynx must be considered. If interstitial emphysema of the neck appears alone or in conjunction with the changes indicated, the diagnosis is even more certain. Local conditions that might cause difficulty in diagnosis are tuberculous cervical lymphadenitis and tuberculosis of the thyroid gland with or without secondary pyogenic infection. The presence of interstitial emphysema might, of course, be due to rupture of pulmonary alveoli with extension to the face and neck. In the last analysis, an accurate diagnosis must rest on a careful laryngoscopic examination, and it must be borne in mind that the anterior surface of the larynx in the median line is the most likely place for perforation to occur, even though the ulceration in this particular region is not extensive.

SUMMARY

Seven cases of perforation of the larynx by a tuberculous process reported from 1836 to 1899 are reviewed. Six cases reported in the same period, in which perforation of the larynx took place by a process that was probably tuberculous, but in which the evidence was not sufficiently conclusive to warrant a definite statement, are also reviewed. One case of definite tuberculous perforation of the larynx is reported from the New Haven Hospital. The latter is the only case reported since 1899, and the probable explanations of this are pointed out. The diagnostic points in tuberculous perforation of the larynx are discussed and are correlated with the pathologic observations.

DENTIGEROUS CYST OF THE ANTRUM OF HIGHMORE WITH UNUSUAL CHARACTERISTICS *

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Cysts of dental origin comprise two primary types: (1) the root or periosteal and (2) the follicular or dentigerous. All varieties of these cysts belong to the group of neoplasms known as odontomas and seem to result from a perverted growth of cells in a tooth follicle. The stimulus which is directly responsible for the growth may be congenital or acquired.

Brief consideration of the embryology of a tooth follicle may throw light on the pathogenesis of these tumors. In the development of a tooth the deeper layer of oral epithelium grows into the mesodermal area of the rudimentary jaw, the projection forming the epithelial cord. Penetrating more deeply into the jaw, the latter divides and becomes the primitive organ which forms, in turn, the enamel tissue. The papilla, a product of the contiguous connective tissue, develops simultaneously with, and invaginates, the enamel organ. These become encircled by a dental sac which arises from the surrounding mesenchyma. The papilla, the enamel organ and the dental sac form the dental anlage or so-called tooth follicle.[1]

ETIOLOGY AND OCCURRENCE

As Wenker [2] observed, the point of special interest in the foregoing connection is that some of the epithelial cells in the enamel organ cease to function, thus becoming a source of the "débris epithéaux dentaries" of Malassez,[3] who in 1885 proposed the theory that all cystic odontomas

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* From the Otolaryngological Service of the Department of Surgery, Lakeside Hospital.

* Read before the Ophthalmological and Oto-Laryngological Section of the Academy of Medicine of Cleveland, Nov. 22, 1929.

1. Lewis, Frederic T., and Stöhr, Philipp: A Text-Book of Histology, ed. 2, Philadelphia, P. Blakiston's Son & Company, 1913.

2. Wenker, R. J.: Follicular or Dentigerous Cyst, J. A. M. A. **77**:1877 (Dec. 10) 1921.

3. Malassez, L.: Sur le rôle des débris épitheliaux paradentaires, Arch. de physiol. norm. et path. **17**:379, 1885.

were derived from this group of cells. Magitot,[4] however, thirteen years previously (1872) published the first important work on the subject, attributing the origin of cystic odontomas to the development of embryonic dental tissue.

The root or periosteal cyst follows inflammatory changes in the root membrane due to disease of, or injury to, the root involved. This type comprises 80 per cent of all cysts and usually involves the upper jaw.

The follicular or dentigerous cyst, with which we are particularly concerned in this paper, comprises 20 per cent of all dental cysts and as a rule involves the lower jaw.[5] Its occurrence in the maxilla is rare. Occasionally, however, it is found even in the gums or orbit. New[6] reported six cases, of which three were in the upper and three in the lower jaw. Usually the cyst is discovered with, or shortly after, the second dentition and in persons between the ages of 20 and 30 years. However, cases in connection with deciduous teeth have been reported in persons as young as 9 years; others with third molar teeth in persons as old as 65 years.

Bland-Sutton[7] held the view that the dentigerous cyst represents an expanded tooth follicle; Dorrance,[8] that it arises from the enamel sac which surrounds an unerupted, supernumerary or abnormal tooth. Most authorities concur in one or both of these views. James H. Harris[9] of the minority, however, expressed the belief that the dental follicle is not a factor in the causation of cysts, but that any pathologic condition affecting Nasmyth's (enamel) membrane may result in noneruption, the internal epithelium of the enamel organ being the source of the cystic fluid. He cited two cases in point. Fisher,[10] by a series of ingenious photomicrographs, furnished "proof" that cyst development is inaugurated by a proliferation of the epithelium due chiefly to inflammatory disturbances or abnormal tissue irritation; and that in follicular

4. Magitot, E.: Mémoire sur les kystes des machoires, Arch. gén. de méd. **2:** 681, 1872.

5. Boyd, William: Surgical Pathology, Philadelphia, W. B. Saunders Company, 1925, pp. 221-222.

6. New, Gordon B.: Cystic Odontomas, Collected Papers of the Mayo Clinic **6:**416, 1914.

7. Bland-Sutton, John: Tumors, Innocent and Malignant, ed. 6, London, Cassell & Company, 1917, pp. 247-249.

8. Dorrance, G. M.: Etiology, Pathology and Treatment of Cysts of the Jaws, J. A. M. A. **77:**1883 (Dec. 10) 1921.

9. Harris, J. H.: Pathology of Tooth-Containing Cysts, and Its Bearing on Eruption, Brit. Dent. J. **35:**1, 1914.

10. Fisher, Guido: Experimental Researches on the Development of Follicular Cysts, Dental Cosmos **53:**1218, 1911.

or dentigerous cysts the enamel epithelium, in performing its formative function, furnishes the epithelial lining of the cysts, their growth being due to diffusion of inflammatory exudate from adjacent parts.

COURSE AND CHARACTERISTICS

Filled with fluid, the cyst progressively expands, sometimes over a long period of years,[11] with consequent absorption of bone and new growth of surrounding tissue.[12] The spongiosa and contiguous tooth roots may yield by absorption and displacement, while the cortex assumes a characteristic parchment-like consistency.

The dentigerous cyst of the antrum of Highmore is unilocular and may be as large as a hen's egg. It is composed of a more or less thick wall of connective tissue irregular in outline. As a rule, it is lined with pavement epithelium, but occasionally it is lined with epithelium of the cylindric type. The cavity may contain serous, seromucous or serosanguineous fluid with degenerated cells and cholesterol crystals. From the wall of the cyst, teeth—rudimentary and misshapen, normal and unerupted or even supernumerary—may project their free ends into the cyst cavity. They rarely occur in great numbers,[13] though Hildebrand[14] found 150 in one cyst. Scudder[15] contended that a true follicular cyst may not necessarily contain a tooth, especially if the cyst began its growth early in relation to the development of the tooth involved. If the cyst contains numerous teeth, the supposition is that multiple supernumerary dental anlagen were present.

The position of the teeth in the cyst varies. If only one tooth is involved, it is commonly well developed and lies more or less in the direction of the contiguous teeth. Impacted teeth may lie in any position in which such teeth are found under other conditions; if several teeth are involved, they are more frequently irregular in position and poorly developed. Retention of developed teeth, however, does not necessarily produce cyst formation,[16] as was shown by Hildebrand when he removed from the jaws of a boy during second dentition over 200 perfectly developed teeth without any evidence of cystic formation. Not infrequently,

11. Goodyear, H. M.: Follicular Dental Cyst, Laryngoscope **38**:328 (May) 1928.

12. Coates, G. M.: Dentigerous Cysts, Prog. Med. **1**:259 (March) 1923.

13. Kaufmann, Eduard: Lehrbuch der speziellen pathologischen Anatomie für Studierende und Aerzte, ed. 6, Berlin, G. Reimer, 1911.

14. Hildebrand, O.: Weiterer Beitrag zur Lehre von den durch abnorme Zahn-Entwicklung bedingten Kiepertumoren, Deutsche Ztschr. f. Chir. **35**:604, 1893.

15. Scudder, C. L.: Tumors of the Jaws, Philadelphia, W. B. Saunders Company, 1912, pp. 197-237.

16. Cumston, C. G.: Dentigerous Cysts, Internat. Dent. J. **25**:256, 1904.

too, teeth impacted concomitantly with cystic formation remain undeveloped because their nourishment is cut off by pressure of the cyst on the nutrient blood vessels.[17]

DIFFERENTIAL DIAGNOSIS

The history, together with clinical and roentgen signs, usually clears up the diagnosis. Given a slow-growing tumor without appreciable pain and associated with an unerupted tooth in either mandible or maxilla, the probabilities favor the presence of a dentigerous cyst. If no tooth is present, the cyst must be differentiated from a root cyst. Usually the history, or a test for pulp vitality, will clear up this question, though frequently only operative procedure will determine the correct diagnosis. In case of an inflammatory or painful cyst, diseased teeth or a recent accident must be suspected. When there is rapid growth and pain not explained by injury or localized infection, a malignant condition should be excluded. In this connection it is well to bear in mind the general constitutional symptoms, together with the spontaneous loss of teeth, fetid and blood-stained nasal secretions and enlargement of cervical lymph nodes, all of which may be found with the rapidly expanding mass of a malignant tumor.[18]

Empyema of the maxillary sinus is usually ushered in with constitutional and other symptoms not seen in a dentigerous cyst; that is to say, there may be typical headache and an elevation of temperature, lack of bulging of sinus walls or fistula formation, presence of purulent discharge and frequently polypi demonstrable in the nasal passages.

On the other hand, dentigerous cysts of the antrum, if uninfected, give no appreciable constitutional reaction. The patient may experience over a certain period a sense of fulness which gradually assumes pressure symptoms, with a slight but noticeable asymmetry of the face. Dilatation of the anterior sinus wall, its parchment-like feeling and springiness and the inner displacement of the lateral nasal wall may clear up the diagnosis. Occasionally, the palate is depressed by the cyst, and a suspected unerupted tooth is absent from the dental arch. If the cyst has ruptured, there may be a fistula communicating with nose or mouth, usually in the lower edge of the canine fossa.

The roentgenogram is an indispensable adjunct to the differential diagnosis of antral conditions. Both Cooper[19] and Feldman[20] have

17. Denney, R. E.: Follicular Dental Cyst, Dental Cosmos **60**:319, 1918.

18. Skillern, R. H.: The Accessory Sinuses of the Nose, ed. 4, Philadelphia, J. B. Lippincott Company, 1923, pp. 154-155.

19. Cooper, W. A.: Dentigerous Cyst in Connection with Third Mandibular Molar, Brit. Dent. J. **40**:410, 1919.

20. Feldman, M. H.: Definite Localization of Supernumerary Tooth Follicle in the Maxillary Sinus, Internat. J. Orthodontia **15**:603, 1929.

demonstrated its value in the definite localization of teeth, the latter being able to locate a supernumerary tooth follicle in the maxillary sinus.

Needle puncture of the antrum, aspiration of the cystic fluid and injection of iodized poppy seed oil 40 per cent with roentgen study will give valuable information as to the contents of the sinus and the shape and size of the cystic mass.

TREATMENT AND PROGNOSIS

As to treatment, nothing but a radical operation will prove of the slightest benefit in these cases. Freudenthal [21] reported the case of a woman 30 years of age whose dentist had drilled a hole in the superior maxilla and irrigated the supposed antrum more or less continuously for two years without relief. She had had a cold and empyema of six years' duration. A radical operation was finally performed by a rhinologist, a dentigerous cyst removed and complete recovery noted in two weeks.

Leon Harris [22] stated that with incision and drainage of a dental cyst, recurrence is inevitable; for, while the swelling subsides after the fluid has been evacuated, the cyst membrane in time secretes and refills the sac. If the tooth follicle is removed, the cyst will never recur. It is also interesting to note, as Dorrance pointed out, that after a radical operation that part of the hard palate which has been involved and thinned by the distention of the cyst returns to its normal contour with deposit of new bone.

Either the Caldwell-Luc or the Denker operation is the one of choice. The prognosis is excellent, as will be seen from the following case.

REPORT OF A CASE

History.—Miss N. B., aged 28, whose family and personal history were unimportant, complained of a sense of fulness in the region of her left antrum and a nasal discharge of several days' duration. She had been susceptible to "head colds" for two years, and three months before admission to the hospital had observed a slight fulness of her cheek over the left canine fossa.

The left canine tooth was missing, all the other teeth of a normal set being present or accounted for. These appeared to be in splendid condition, and the gums and buccal mucosa were normal. There was a slight fulness of the anterior wall of the left sinus, causing a moderate asymmetry of the face. Palpation of the tumor elicited a parchment-like crackle and springiness over this wall. No fistula was discovered, but there was a distinct bulging mass, the size of the end of the thumb, easily seen in the left anterior nares immediately beneath the inferior turbinate, which displaced both the turbinate and the lateral nasal wall medially (fig. 1).

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21. Freudenthal, W.: Dentigerous Cyst of Antrum of Highmore, Am. Med. **6**:277, 1911.

22. Harris, Leon: Dental Cysts, Dental Cosmos **64**:1165, 1922.

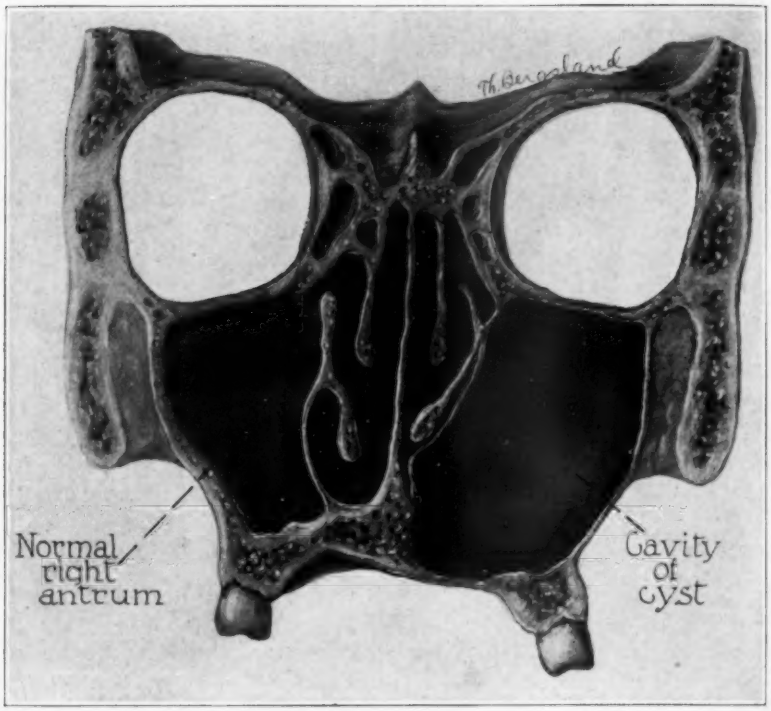

Fig. 1.—Normal right antrum and cavity of cyst. Note the thinning of antral wall about the cyst, the depression of the palate together with compression of the lateral wall and elevation of the floor of the nose. The inferior turbinate is displaced medially and there is a distinct bulging of the cyst beneath its inferior aspect.

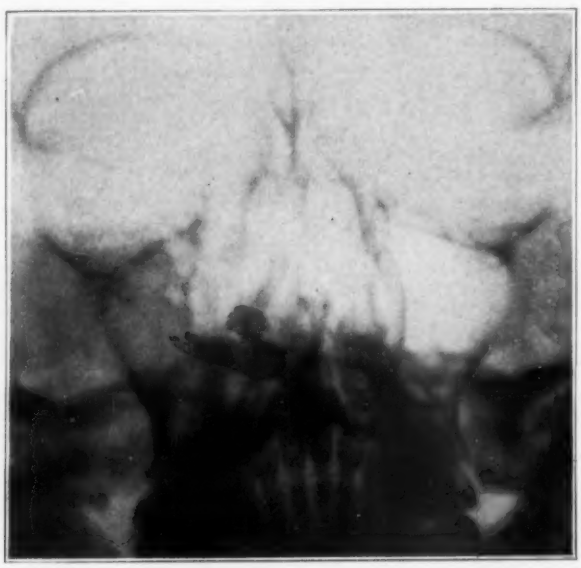

Fig. 2.—Postero-anterior view of cystic antrum before operation. The supernumerary molar and canine teeth are easily demonstrable.

A tentative diagnosis of dentigerous cyst was made. Roentgenograms confirmed this diagnosis and demonstrated what appeared to be two teeth, one in the lower anteromedial portion and the other in the anterocentral portion of the cyst (fig. 2).

The day before operation a needle puncture of the cyst wall was done beneath the inferior left turbinate, and about 10 cc. of yellowish-chocolate-colored odorless fluid was aspirated. Through the same needle, 8 cc. of iodized oil was injected. Roentgenograms showed an unusually extensive cyst, kidney-shaped and unilocular, apparently filling the entire antrum (fig. 3).

Operation.—With the patient under local anesthesia, a Caldwell-Luc operation was performed. There was a large dehiscence in the maxillary wall just above the alveolar process. This was divided by a bridge of bone containing the left

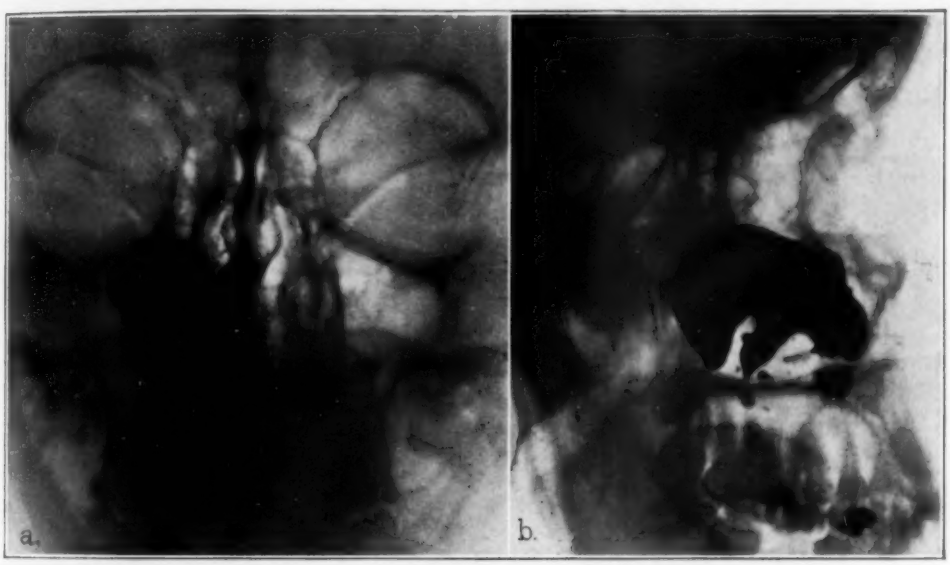

Fig. 3.—Postero-anterior (*a*) and lateral (*b*) views of cyst after injection of iodized oil. Note the great size and irregular contour. It is unilocular and almost completely fills the antrum.

canine tooth of the second dentition. It grew in a downward and forward direction, but was entirely external to the cyst itself. This tooth, of almost normal development, was found to have a fissure 0.3 mm. wide and about 2 mm. in depth across the root on its posterolateral aspect, presumably the impression of a sharp shelf of bone. (Feldman [20] pointed out that impacted teeth are frequently caught in such a manner in the maxillary sinus.) With wide exposure, the cyst, occupying the entire cavity of the antrum, was dissected free from the bone intact, and a large second dehiscence was found in the outer nasal wall. The cyst contained about 20 cc. of a yellowish-chocolate-colored fluid, as well as a poorly developed supernumerary molar tooth attached to the wall of the cyst at the nasofacial angle. The occlusal surface projected into the cyst cavity (figs. 4 and 5).

Subsequent to the operation there was moderate bleeding, but this was readily controlled with epinephrine packs in the nares. After six days, the left cheek

became markedly edematous, and the left eye completely closed. With cold compresses to the face, this condition subsided in three days. The highest temperature reached was 38 C. The serosanguineous discharge from the antrum persisted slightly for about three weeks, but gradually decreased until complete recovery occurred.

Fig. 4.—Sac of the cyst laid open, showing its inner wall.

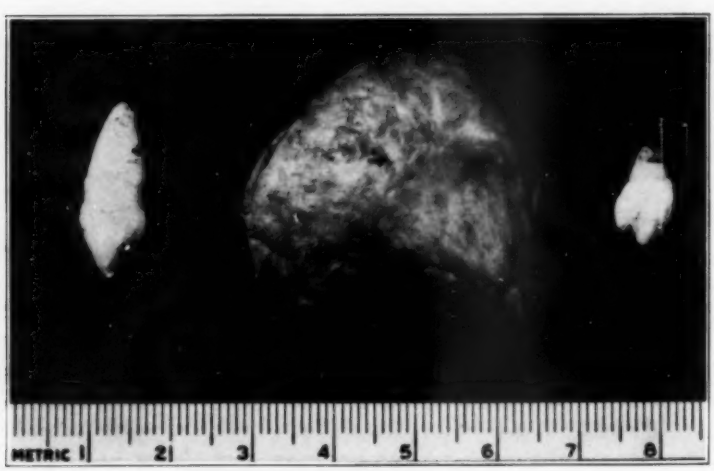

Fig. 5.—External view of the cyst together with the impacted canine and the supernumerary molar, found at operation. The latter projected from the wall of the cyst into its cavity. Note the size of the cyst and the fissure in the side of the root of the canine tooth.

Pathologic Report.—The gross specimen consisted of a roughly ovoid, thin-walled cyst and two teeth, one of which was apparently a canine, while the other appeared incompletely and atypically formed and was not identified. The cyst

bore a striking resemblance in outline to an infant's stomach, and there was a broad, thin line of attachment along what would correspond to the lesser curvature. The cyst measured 3.5 cm. in length by 1.3 cm. in thickness. The wall was fibrous, and the outer surface was smooth. The lining was roughened by what appeared grossly to be fibrin, and the contents had previously been evacuated.

The impression gained from this examination was that the condition was a dentigerous cyst.

Sections through the cyst wall included a thin but continuous lining membrane of stratified squamous epithelium. The epithelium varied in thickness and in some areas appeared transitional. The wall of the cyst was fibrous and of loose texture. Immediately beneath the epithelium there were a number of collections of what appeared to be hemosiderin, some of which was extracellular and some of which was deposited in large mononuclear phagocytes. The entire wall was diffusely infiltrated by polymorphonuclear leukocytes, endothelial cells and lymphocytes. The first predominated. There were a number of small hemorrhagic areas, and these were most commonly noted in regions in which the wall was occupied by what appeared to be well vascularized granulation tissue. The diagnosis was dentigerous cyst, the seat of subacute inflammation.

CONCLUSIONS

A case of dentigerous cyst is reported for the reason that it is not only rare in type but unusual in several of its characteristics and relations.

1. A unilocular cyst completely filled the antrum, and, despite its great size, had formed no fistulous tract into either mouth or nose. There were, however, two large bony dehiscences.

2. It contained a poorly developed supernumerary molar, indicating that the cyst had taken its origin from an aberrant tooth bud or so-called follicle.

3. The cyst bore no direct relation to the left canine tooth, which was unerupted and missing from the arch. This tooth, anchored by a shelf of bone, lay wholly outside the cyst.

4. This cyst was of exceptionally long growth, as evidenced by its size and the impacted canine, which normally erupts between the tenth and thirteenth years of life. It may be concluded, therefore, that the shelf of bone which anchored the tooth so securely was gradually driven into the side of its root by the slowly forming cyst over a long period of years.

LATERAL TRANSTHYROID PHARYNGOTOMY

TROTTER'S OPERATION FOR MALIGNANT CONDITIONS OF THE LARYNGOPHARYNX *

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Drawbacks to operations on the laryngopharynx may be classified, according to Trotter,[1] into three groups: (1) the uncertainty of cure, which is the most important; (2) the danger of the operation and the distresses of convalescence, and (3) the mutilation and disability that may be left by an operation otherwise perfectly successful. This still holds true.

Intrinsic cancer is being well attacked by both laryngofissure and laryngectomy by Fellows of the American Laryngological Association. But I think that something can be done for patients who are suffering from so-called extrinsic cancer of the larynx. Better terms are epilaryngeal cancer, a growth originating on the epiglottis, aryepiglottic folds, pyriform sinus or lateral wall of the pharynx, and hypopharyngeal cancer, occurring in the lower portion of the pharynx.

In this presentation I claim nothing original, f[illegible] quote freely from Mr. Trotter's articles and personal communicatio[illegible]t by plaster casts and photographs, I shall attempt to show a proce[illegible]ure by which to attack malignant conditions of the pharynx: lateral tran[illegible]pharyngotomy. I shall limit myself to that portion known as th[illegible]ngopharynx.

A few years ago, while visiting in England, I saw Mr. Trotter with comparative ease remove by this route a postcricoid carcinoma, an operation in which heretofore the larynx as well has been sacr[illegible]ced. I became enthusiastic over it, and since that time I have had occasion to perform this operation on nine patients. If my results ar[illegible]t go[illegible] is due to the operator and not to the operation. In these nine cases I had an operative mortality of two, or 22.3 per cent, both of the patients dying within

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* Submitted for publication, June 21, 1930.

* Read before the American Laryngological Association, Swampscott, Mass., May 21, 1930.

* The dissections and casts were made with the assistance of Dr. H. P. Mosher in his laboratory at the Harvard University Medical School in February, 1930.

1. Trotter, Wilfred: Principles and Technique of the Operative Treatment of Malignant Disease of the Mouth and Pharynx, Lancet **1**:1075 (April 19) 1913; 1147 (April 26) 1913.

thirty-six hours of septic pneumonia. Three patients had a recurrence in the glands six months, and one patient nine months, following operation; one lived for one and a half years, and two are living at this writing, one patient now for over one year and the other for eight months.

Microscopically I am concerned only with the squamous cell epithelioma. Mr. Trotter's classification shows (fig. 1 *A*) the hypopharyngeal group which involves the cricoid region and the posterior pharyngeal wall, and which is practically limited to women. Three of my cases belonged to this group.

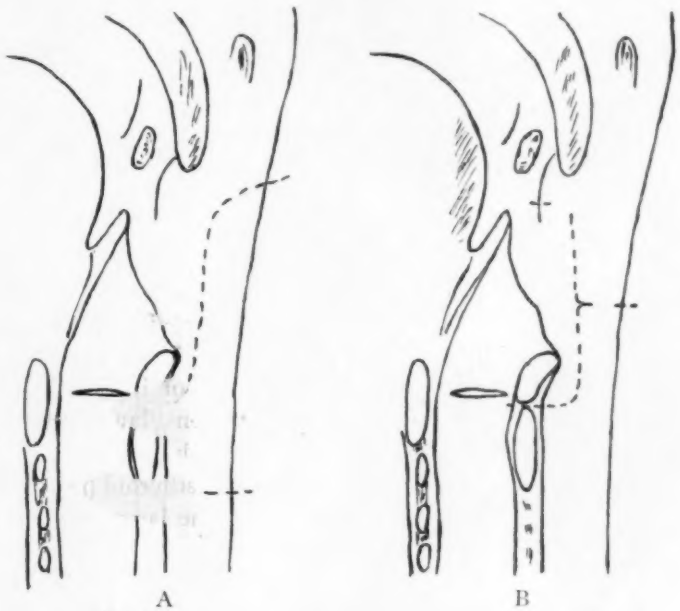

Fig. 1.—*A*, the hypopharyngeal group, practically limited to women (Wilfred Trotter); *B*, the epilaryngeal group, the type ordinarily occurring in men (Wilfred Trotter).

Figure 1 *B* shows the group known as the epilaryngeal, which involves the arytenoids, aryepiglottic folds, epiglottis, pyriform sinus and possibly the base of the tongue. This group is almost entirely limited to men. Six of my cases belonged to this group.

In the order named, a growth involving the epiglottis, aryepiglottic folds, lateral wall of the pharynx, postcricoid and pyriform sinus gives the best prognosis. If the growth has had its origin from the lateral wall of the larynx, it soon involves the pyriform sinus and thyroid cartilage, and nothing short of complete laryngectomy, with a partial or entire cervical esophagus, will be of any use. This group is the most

discouraging one for any operative procedure, yet if the condition is seen early enough, a few additional years of life may result from the operation. By the removal of the shaded portion of the hyoid bone and thyroid cartilage one has an approach to these conditions (fig. 3).

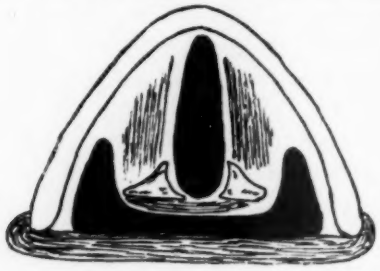

Fig. 2.—Diagram of section through the pyriform sinus of the pharynx to show its relation externally to the thyroid cartilage and internally to the lateral wall of the larynx (Wilfred Trotter).

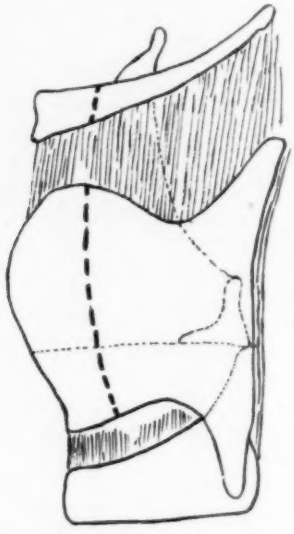

Fig. 3.—The finely dotted lines indicate the relative positions of the aryepiglottic fold, the arytenoid cartilage and the vocal cord. The thickly dotted lines show where the great cornu and the thyroid ala are divided in the operation. When these parts have been removed, the laryngopharynx is accessible (Wilfred Trotter).

After a preliminary tracheotomy, the incision for ordinary transthyroid pharyngotomy would be along the anterior border of the sternomastoid muscle, reflecting anteriorly and posteriorly the muscles of the larynx. But as most of these patients, by the time they are examined,

have enlarged glands of the neck, from either infection or lymphatic extension, it becomes necessary to perform a resection, and the former incision is inadequate. We then make an additional incision more or less at right angles to the original one, running from the angle of the lower jaw downward, forward and upward to the symphysis (fig. 4).

These flaps and the platysma are turned back; the superficial fascia, the submaxillary gland and the deep fascia are likewise removed upward and backward as far as the spinal accessory nerve and posterior triangle.

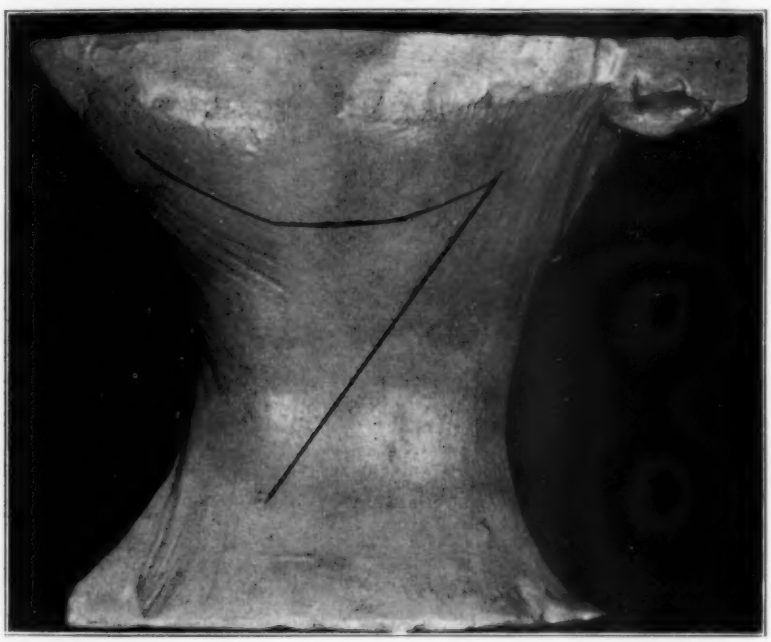

Fig. 4.—A preliminary tracheotomy having been performed, an incision is made along the anterior edge of the sternomastoid from the angle of the jaw to the suprasternal notch; from this, another incision more or less at right angles extending along the lower border of the jaw to the symphysis. These incisions vary, depending on the type of operation and the amount of tissue to be removed.

This step exposes the deep vessels of the neck and the muscles surrounding the larynx (fig. 5).

All the vessels that come off anteriorly from the great vessels are ligated and cut. The internal jugular is ligated and resected. The external carotid may or may not be ligated. The sternomastoid may or may not be removed, depending on the glandular involvement (fig. 6).

The next step is to suture the sternomastoid to the prevertebral fascia; this covers the great vessels and thus protects them from infec-

tion when the pharynx is opened. The area is drained by a counter-opening posteriorly (fig. 7).

The following muscles are then reflected from the hyoid bone and thyroid cartilage: the hypoglossal, thyrohyoid, sternohyoid, sterno-thyroid, stylopharyngeus, and the inferior and middle constrictors of the pharynx. One is then ready to remove, by means of heavy scissors, the exposed portions of the hyoid bone and the greater portion of the thyroid cartilage (fig. 8).

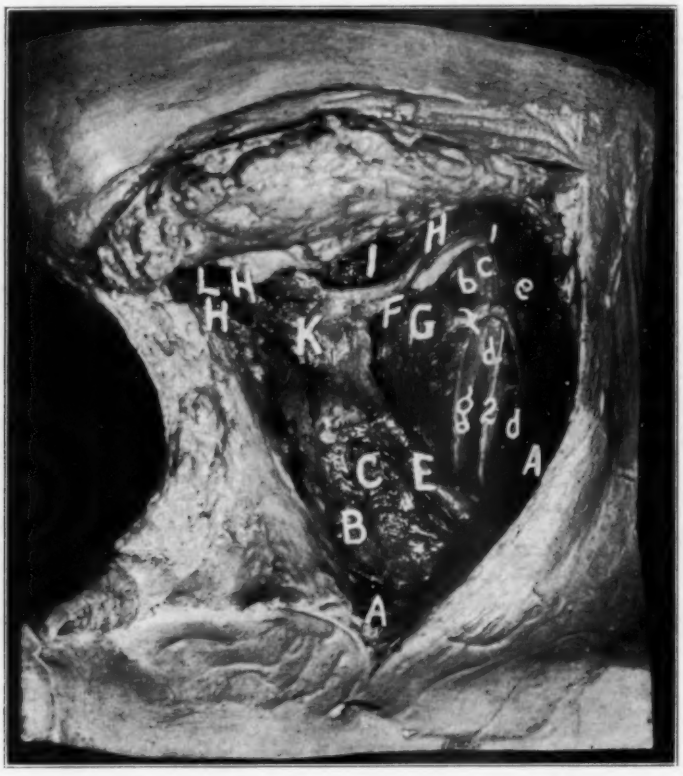

Fig. 5.—This shows the skin and platysma retracted. The cervical fascia, superficial lymphatic glands and submaxillary gland are removed. The anterior edge of the sternomastoid has been retracted outward to expose the structures lying beneath it. The carotid sheath is opened, showing the vessels. The muscles shown are: *A*, the sternomastoid; *B*, the sternohyoid; *C*, the sternothyroid; *D*, the thyrohyoid; *E*, the omohyoid; *F*, the stylohyoid (cut); *G*, the hyoglossus; *H*, the digastric; *I*, the submaxillary fossa; *K*, the suprahyoid aponeurosis; *L*, the mylohyoid, and *Z*, the pomum adami. The vessels are: *a*, the common carotid; *b*, the external carotid; *c*, the internal carotid; *d*, the internal jugular vein; *e*, the anterior division of the temporomaxillary vein joining the facial; *f*, the facial, and *g*, the carotid sheath. The nerves are: (1) the hypoglossal and (2) the descendens noni.

This permits one to palpate the growth through the pharyngeal aponeurosis and mucous membrane, and to decide where to make the opening into the pharynx so as to be well around the malignant area; or if the growth is too large to remove, one may back out, so to speak, and not proceed any further (fig. 9).

The next step is to open the pharynx, an incision which gives an approach to either a hypopharyngeal or an epilaryngeal growth, and which may be continued upward by going through the mandible, as in one of my cases, to remove a growth higher up (fig. 10).

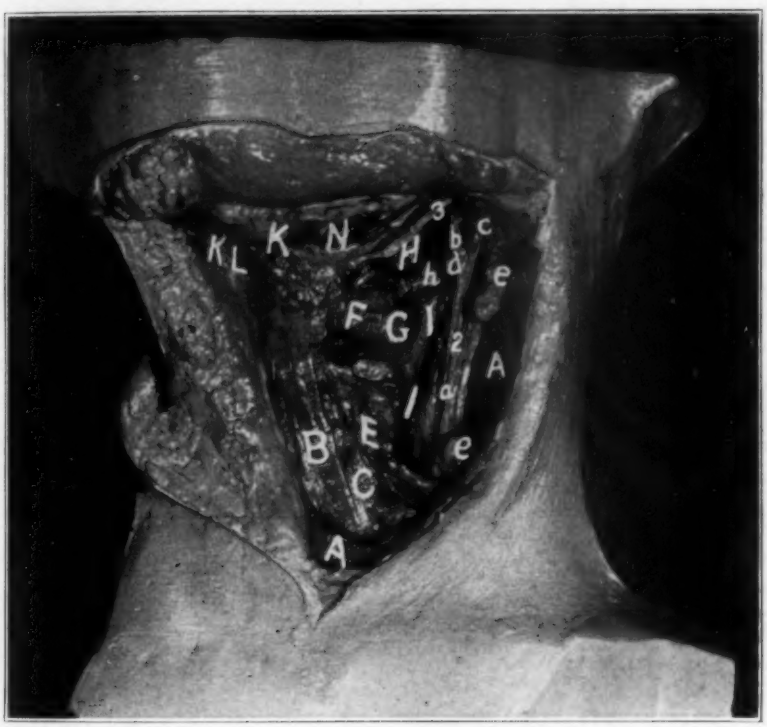

Fig. 6.—This shows the deep cervical fascia removed and the internal jugular vein ligated and resected. All veins and arteries coming off anteriorly have been ligated and cut. The pneumogastric nerve is seen. The thyrohyoid muscle is cut, exposing the thyrohyoid membrane. The inferior constrictor has been separated from the thyroid cartilage. The hyoid bone is also exposed. The muscles shown are: *A*, the sternomastoid; *B*, the sternohyoid; *C*, the sternothyroid; *E*, the omohyoid; *F*, the thyrohyoid (cut); *G*, the thyrohyoid membrane; *H*, the hyoglossus; *I*, the inferior constrictor; *K*, the digastric; *L*, the mylohyoid; *M*, the suprahyoid aponeurosis and *N*, the submaxillary fossa. The vessels are: *a*, the common carotid; *b*, the external carotid; *c*, the internal carotid; *d*, the superior laryngeal; *e*, the internal jugular ligated and resected; *f*, the thyroid cartilage-cornua, and *h*, the hyoid bone. The nerves are: (1) the pneumogastric; (2) the descendens noni and (3) the hypoglossal.

Before the operation, one has a fair idea of how much tissue it will be necessary to remove, and the flap is made sufficiently large to fill up the defect. The picture shows a flap of skin turned in to make up a defect in a postcricoid carcinoma. A feeding tube is inserted into the esophagus. Later, a plastic operation is performed to close the neck (fig. 11).

As illustrated in figure 12 *A*, if the growth is on the anterior wall of the esophagus, the flap will be made from *A* to *B*. The black portion

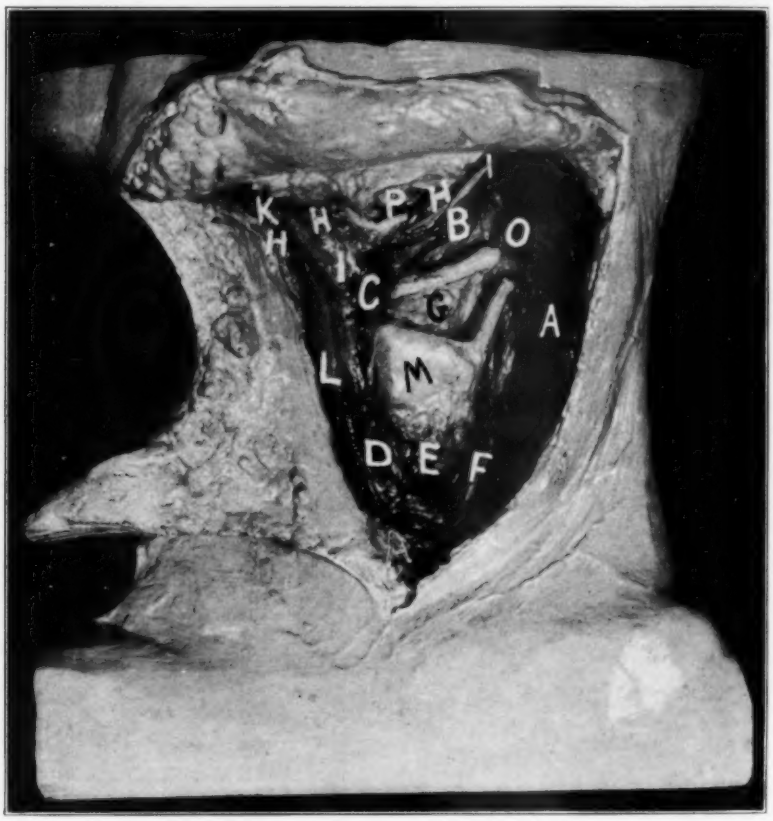

Fig. 7.—This shows the anterior edge of the sternomastoid sutured to the prevertebral fascia covering and protecting the great vessels. The area is drained by a counteropening posteriorly. The muscles have been reflected from the hyoid bone and the thyroid cartilage. The constrictors of the pharynx are reflected, and the great cornu of the hyoid bone and thyroid cartilage exposed for removal. The muscles are: *A*, the sternomastoid sutured to the prevertebral fascia; *B*, the hyoglossus; *C*, the thyrohyoid; *D*, the sternothyroid; *E*, the cricothyroid; *F*, the inferior constrictor; *G*, the thyrohyoid membrane; *H*, the digastric; *I*, the suprahyoid aponeurosis; *K*, the mylohyoid; *L*, the sternohyoid; *M*, the thyroid cartilage; *N*, the great cornu of the hyoid; *O*, the middle constrictor, and *P*, the submaxillary fossa. The nerve shown (1) is the hypoglossal.

is removed and then the flap is turned in from *B*. If the growth occurs posteriorly, the flap is turned in from *B* over the muscles and vessels, as shown in figure 12 *B*.

REPORT OF CASES

CASE 1.—M. T., a white woman, aged 60, married, was referred to me on March 30, 1928, for difficulty in swallowing. Nothing in the family history was of any importance. Her general health had been good until September, 1927,

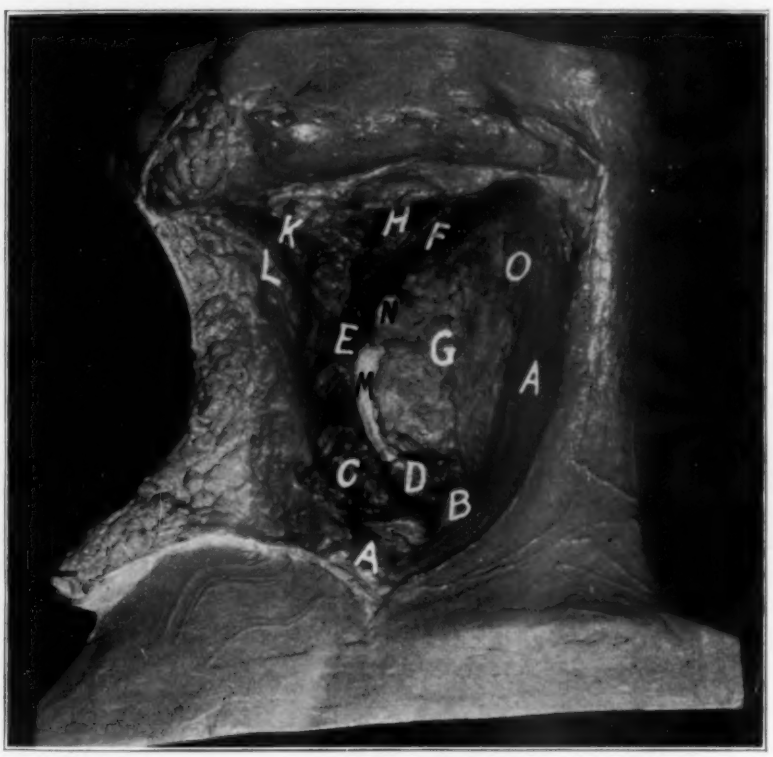

Fig. 8.—This shows the great cornu of the hyoid bone removed as well as the greater half of the thyroid cartilage; the wall of the pharynx is now well exposed and ready for incision. The muscles and structures shown are: *A*, the sternomastoid; *B*, the inferior constrictor; *C*, the sternothyroid; *D*, the cricothyroid; *E*, the thyrohyoid; *F*, the hyoglossus; *G*, the pharynx ready to be opened; *H*, the submaxillary fossa; *K*, the digastric; *L*, the mylohyoid; *M*, the thyroid cartilage removed; *N*, the great cornu of the hyoid removed, and *O*, the middle constrictor.

when she had trouble with swallowing, without pain. She had a slightly enlarged thyroid gland, for which she had been treated at one of the local hospitals; at another hospital she had had her tonsils removed. The only other complaint that

she had besides slight trouble in swallowing was the expectoration of considerable phlegm which accumulated in her mouth. The Wasserman test, urinalysis, roentgen examination of the chest and blood chemistry all gave negative results.

Laryngeal examination showed the left cord and arytenoid fixed and a mass in the postcricoid region, involving the entire hypopharynx. The clinical diagnosis was carcinoma, and an operation was suggested.

I did not see this patient again until November, 1928, ten months later, when she was admitted to the City Hospital. There had been a gradual increase of difficulty in swallowing but no pain; the patient stated that she had had a number

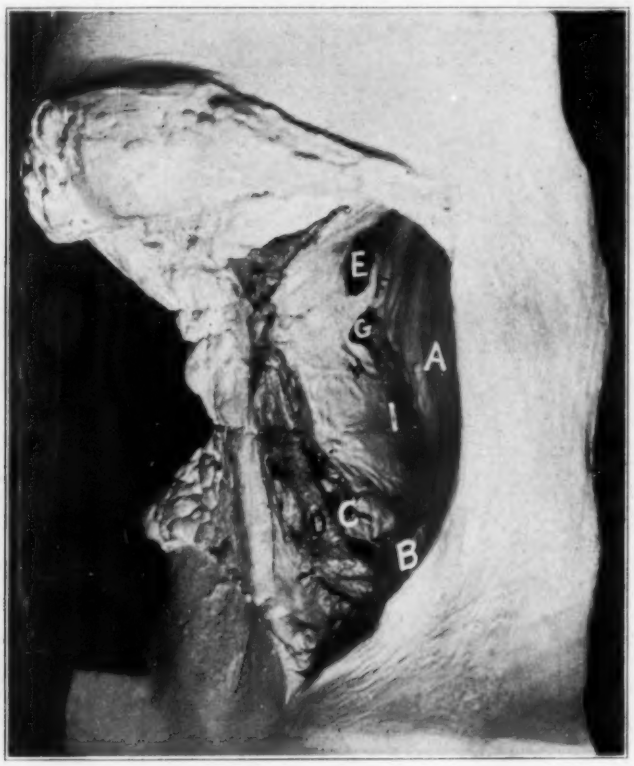

Fig. 9.—This shows the pharynx opened, exposing the base of the tongue, epiglottis, arytenoids and postcricoid regions. This gives an excellent approach to a growth in either the epilarynx or the hypopharynx. The muscles and structures shown are: *A*, the posterior wall of the pharynx; *B*, the sternomastoid; *C*, the cricothyroid; *D*, the sternothyroid; *E*, the base of the tongue; *F*, the epiglottis; *G*, opening into the larynx; *H*, the arytenoids, and *I*, the cricoid.

of roentgen treatments. She had had no difficulty in breathing. She had lost about 50 pounds (22.7 Kg.) in the previous four months. Laryngeal examination, on November 1, showed an extensive growth which seemed to spring from the posterior wall of the esophagus and which extended well into the pyriform

sinus; both arytenoids were edematous. This growth apparently had progressed considerably in ten months. The patient and her family wanted something done, although I could promise them no hope.

On November 2, I performed a transthyroid pharyngotomy. A tracheotomy was performed and a no. 4 tracheotomy tube was inserted. An incision about 3 inches (7.6 cm.) long was made across the median line of the neck; then an incision was made from the upper portion to the angle of the jaw and a lower incision to the clavicle. The skin was reflected backward, the fascia to the posterior border of the sternomastoid was removed along with the resection of the internal jugular; the external carotid was ligated, and the submaxillary gland on

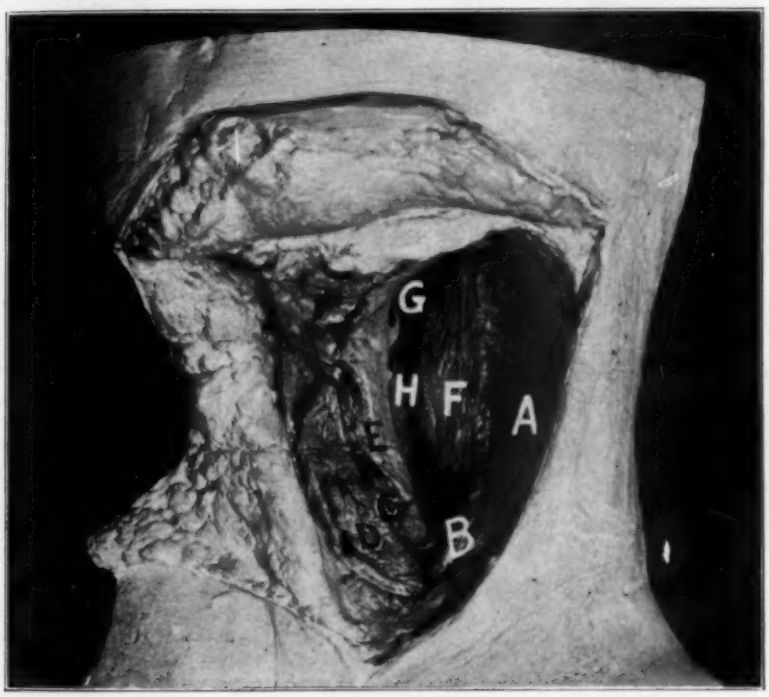

Fig. 10.—The muscles and structures shown are: *A*, the sternomastoid; *B*, the inferior constrictor; *C*, the cricothyroid; *D*, the sternothyroid; *E*, the cricoid; *F*, the postpharyngeal wall; *G*, the epiglottis, and *H*, the arytenoids.

the left side was removed. The left side of the thyroid cartilage was removed. The sternomastoid was sutured to the prevertebral fascia. The inferior constrictor was cut through, exposing the lateral wall of the pharynx; this was opened and the growth inspected. The skin was reflected into the pharynx; the upper part of the flap was sewed to the pharyngeal mucous membrane. The same procedure was followed below. The feeding tube was passed into the esophagus and anchored at the opening of the neck. A dressing was applied. The patient died on the fourth day following operation. The pathologic report showed carcinoma.

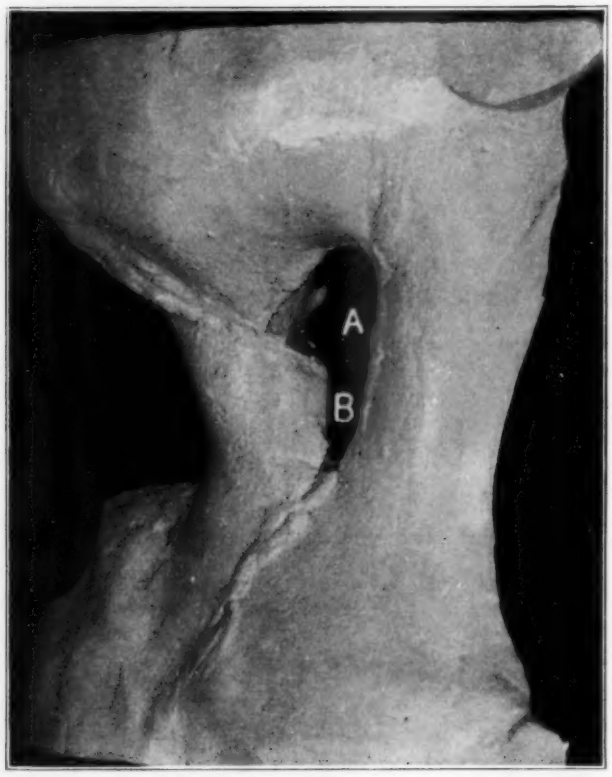

Fig. 11.—This shows the closure for postcricoid growth, when it has become necessary to turn in a flap of skin from the neck to make good the defect in the pharynx. This gutter is closed later by a plastic flap. *A*, the posterior pharyngeal wall, and *B*, the skin flap turned in.

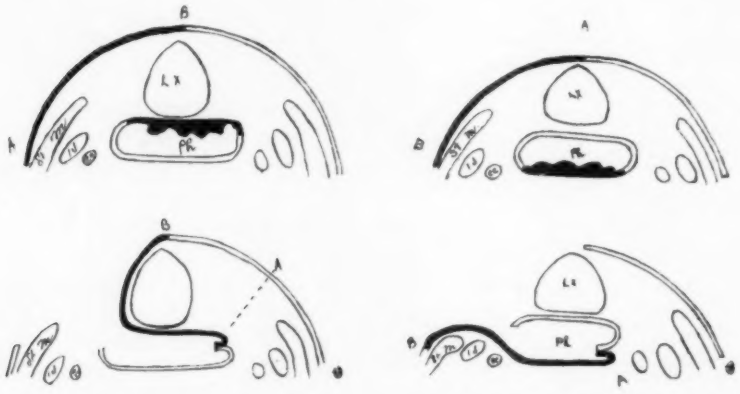

Fig. 12.—*A*, disposition of skin flap in operation for removal of a hypopharyngeal growth-tumor on the anterior wall (Wilfred Trotter); *B*, disposition of skin flap in operation for removal of a hypopharyngeal growth-tumor on the posterior wall (Wilfred Trotter).

CASE 2.—S. K., a white woman, aged 57, married, consulted me on Jan. 17, 1929, complaining of inability to swallow. Her father died of cancer, and her husband died of some internal growth (?). The personal history was negative. The present illness began in August, 1928, with difficulty in swallowing. She noticed some enlarged glands on the side of the neck which at times were painful, the pain radiating to the right ear. Laryngeal examination showed the left cord abducting and adducting normally, with a mass in the postcricoid region. The right cord was interfered with somewhat in these movements by a mass in the right aryepiglottic fold and posterior lateral wall of the pharynx. Urinalysis, the Wassermann test, blood chemistry and roentgen examination of the chest gave negative results.

An operation was performed on Jan. 28, 1929. A tracheotomy was performed under procaine hydrochloride anesthesia, followed by intratracheal anesthesia. An incision was made so as to cover in the defect caused by the removal of a growth from the posterior wall of the larynx. The fascia and glands along the deep vessel were removed. The internal jugular was ligated and resected. The submaxillary gland was removed. The muscles of the larynx and the hyoid bone were freed; the hyoid bone and a portion of the thyroid cartilage were removed, exposing the wall of the pharynx. The growth was then palpated and found to extend from above the larynx to below the third ring of the trachea. Because of this extension, it was decided not to enter the pharynx. On February 4, gastrostomy was performed. The patient refused further operation, and left the hospital in February. She died on June 9. The pathologic report on a specimen showed it to be undifferentiated carcinoma of the esophagus.

CASE 3.—H. B., a white man, aged 56, married, was referred to me in June, 1929. The personal history was negative. The patient was well until three months before examination, when he noticed a small ulceration on the buccal surface of the right cheek. This area became more and more eroded, until it reached the size of a quarter. It then involved not only the buccal mucous membrane, but also the jaw and the side of the mouth. This became so painful that the patient had to seek medical aid. There were some enlarged cervical glands on the right side. Urinalysis, the Wassermann test, blood chemistry and roentgen examination of the chest gave negative results.

On June 10, under rectal anesthesia, an incision was made in the middle of the chin downward and outward to the middle of the clavicle on the right side. Flaps were turned backward and forward. The glands of the neck, the deep superficial fascia and the submaxillary gland were removed. The external carotid artery was ligated. An incision was then carried upward through the lip, and the entire right side of the face was turned back. The jaw was incised at the ramus, and 3 inches of the right side of the jaw were excised. Then by means of diathermy the right half of the tongue, the floor of the mouth and the side of the pharynx were removed. The patient was discharged from the hospital three weeks later. I did not see him again until October 3, when a slight recurrence on the inner surface of the ramus of the right jaw was observed. He was admitted to the hospital on October 7 for further operation by means of surgical diathermy. While rectal anesthesia was being administered, he suddenly ceased to breathe. The pathologic report showed squamous cell carcinoma.

CASE 4.—J. E., a white man, aged 40, married, was admitted to the hospital on Dec. 16, 1929, with a complaint of difficulty in swallowing. His mother had died of cancer at the age of 58. Nothing in the antecedent personal history was of interest. The patient had had trouble with his throat for a year, and had gradually

encountered difficulty in swallowing. In attempting to swallow food he readily gagged, and he felt that there was an obstruction in his throat. The condition became progressively worse. He complained of slight hoarseness. Laryngeal examination showed a large growth springing from the right side of the pyriform sinus, which caused a sluggish movement of the right arytenoid. Urinalysis, blood chemistry, the Wassermann test and roentgen examination of the chest gave negative results.

An operation was performed on Jan. 6, 1930. A transverse tracheotomy was performed, and then a laryngoscopy for direct inspection. I was under the impression that the growth sprang more from the posterior wall, as a probe would not pass around it. An incision was made from the angle of the jaw downward and forward beyond the median line, then downward over the left side of the thyroid cartilage to just above the tracheotomy opening and then backward beyond the border of the sternomastoid muscle. This flap was dissected backward; the glands of the neck were dissected, and several glands were found along the carotid sheath. The internal jugular vein was ligated and resected. The submaxillary gland was removed along with the deep fascia. The sternomastoid muscle was sutured to the prevertebral fascia protecting the great vessels. The muscles were freed from the hyoid bone and thyroid cartilage, exposing the lateral wall of the pharynx, which was opened; on inspection, the growth proved to be so extensive that it involved the entire pyriform sinus. It was then decided to remove the entire growth and the larynx from above downward, with about 2 inches (5 cm.) of the cervical esophagus. All bleeding was controlled. A skin flap was turned in; the upper part of the esophagus was sutured to the skin and the lower part to the lower portion of the skin flap, thereby forming the posterior wall of a new esophagus. A feeding tube was inserted through the nostril, and the trachea was sutured to the skin. An extensive operative procedure was thus employed. The man died on January 9, three days after operation. The pathologic report showed the specimen removed to be a squamous cell carcinoma, type 4.

CASE 5.—J. D., a white man, aged 54, married, was referred to me on Oct. 12, 1929, complaining of pain on the left side of his tongue.

Laryngeal examination showed a postcricoid carcinoma on the right side. Urinalysis, the Wassermann test, blood chemistry and roentgen examination of the chest yielded negative results.

An operation was performed on November 4. After preliminary tracheotomy, an incision was made so that a flap could be turned in to form the anterior wall of the esophagus, to replace the amount of tissue that was to be removed. The fascia and the glands along the vessels were removed. The jugular vein was ligated and resected. The external carotid was ligated. The submaxillary gland was removed. The sternomastoid was sutured to the prevertebral fascia. The pharynx was exposed but was not opened. The dissection of the gland took a long time because roentgen and radium treatment had fused the various planes, making dissection difficult. The skin was closed with the intention of completing the operation later. The patient did not return for observation as soon as I expected; when seen, he stated that he did not wish to have anything further done. The growth was in the postcricoid region, involving the pyriform sinus. The patient was discharged from the hospital on November 16, but continued to have roentgen and radium treatment. He became worse, and died on March 5, 1930.

A section was made for pathologic examination. The gland was found to be entirely replaced by invading groups of carcinomatous cells and fibrous productive tissue. The carcinomatous cells appeared moderately active, with larger

cells and mitosis present, but there was also considerable evidence of differentiation in the formation of epithelial pearls. There was considerable protective fibrosis. The diagnosis was grade 3 carcinoma in the lymph gland.

CASE 6.—E. W., a white woman, aged 55, married, was referred to me on Oct. 29, 1927, with a complaint of difficulty in swallowing.

Laryngeal examination showed both arytenoids to be slightly edematous. Both cords abducted and adducted normally. On phonation there was seen in the postcricoid region of the esophagus the upper surface of an irregular mass with ulceration in the center; some cervical glands were palpable on the left side.

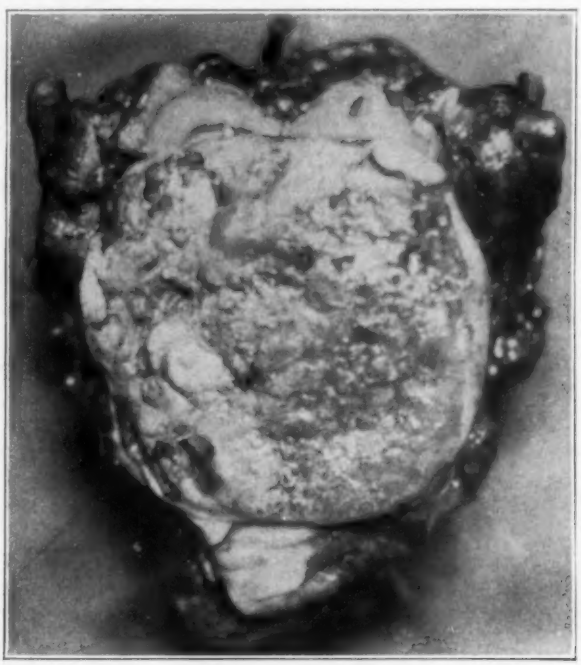

Fig. 13 (case 6).—Squamous cell carcinoma.

Urinalysis, the Wassermann test, blood chemistry and roentgen examination of the chest yielded negative results.

On November 19, the entire mass, including the anterior wall of the esophagus and larynx, was removed with about 2 inches of the posterior wall. The growth was a squamous cell carcinoma. Figure 13 shows the extent of the growth.

On Feb. 14, 1928, a gastrostomy was performed to enable the patient to be fed while we were reconstructing the anterior wall of the esophagus. Figure 14 *A* shows the upper opening and lower opening of the esophagus with the trough forming its posterior wall. After the gastrostomy was performed, the nasal feeding tube was removed.

On March 1, 1928, the edges of this trough were undermined. A flap was turned down from the chin to form the anterior wall. The parts undermined were brought together in the median line; a double row of sutures was made to prevent leakage. Figure 14 *B* shows the patient after operation.

On pathologic examination, no evidence of papillary ingrowth into the tumor area was found. The diagnosis was squamous cell carcinoma.

CASE 7.—P. D., a white man, aged 63, married, was referred to me on March 16, 1929, for a swelling of the neck of three weeks' duration. He had been troubled with occasional hoarseness.

Laryngeal examination showed the right arytenoid and cord moving freely on abduction and adduction; above the left arytenoid there was an irregular mass. Figure 15 shows the extent of the swelling before operation. Urinalysis, the Wassermann test, blood chemistry and roentgen examination of the chest yielded negative results.

An operation was performed on April 8. Under rectal anesthesia a low transverse tracheotomy was performed. An incison was made along the anterior

A B

Fig. 14 (case 6).—*A*, appearance of patient after gastrostomy; *B*, appearance of patient after operation.

border of the sternomastoid muscle from the angle of the jaw downward to the level of the hyoid bone, going beyond the median line of the neck, then downward to within 1 cm. of the tracheotomy wound and then backward to the anterior border of the sternomastoid. The skin flaps turned back exposed the muscles of the entire left side of the neck to the angle of the jaw. The deep fascia of the neck and the internal jugular vein were removed along with the submaxillary gland; this exposed a mass of glands (fig. 16 *A*) below the sternomastoid muscle. This mass involved the deep vessels of the neck. The carotid artery was dissected free from it. The vagus nerve was freed from this mass, and the upper part of the jugular vein was ligated and cut at the angle of the jaw; the muscle, vein and mass of glands were removed. The pharynx was opened, exposing a carcinoma of the posterior part of the larynx which was dissected free and removed. The anterior esophageal wall was sutured to the mucous membrane

of the larynx; the pharynx was closed with three drains. The edges of the incision were brought together by means of clips. The dissection of glands was extensive and adherent to various structures. Exposing the pharynx, a very small postcricoid growth was removed (fig. 16 *B*). There was not much loss of tissue in the esophagus; it was not necessary, therefore, to turn in the flap to make up a defect.

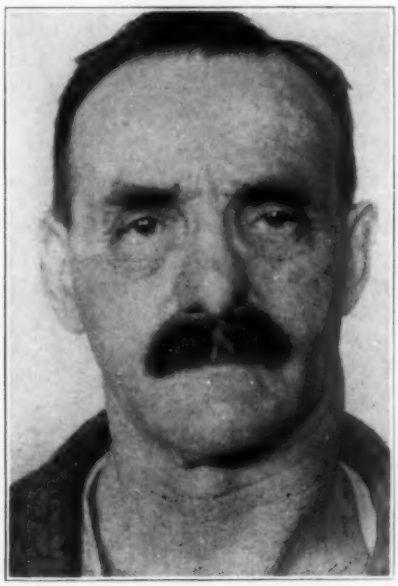

Fig. 15 (case 7).—Swelling of the neck before operation.

A B

Fig. 16 (case 7).—*A*, mass of glands removed; *B*, postcricoid growth.

The patient recovered from his operation, got along without his tracheotomy tube and had a good voice. On August 21, he had a recurrence of the growth in the posterior triangle. Deep roentgen treatment and radium treatment was given without much benefit. The patient died on December 19.

There had been no recurrence of growth within the pharynx. It is quite noticeable that in these cases the recurrences have been in the glands and not at the point of original growth.

Pathologic examination of the tissue on April 17 revealed an invasive growth composed of groups and strands of carcinoma cells. The growth was very cellular, with only slight fibrous production in the stroma. The cells were undifferentiated and showed numerous mitotic forms. The diagnosis was carcinoma. On April 27, examination of a gland in the neck revealed extensive invasion and replacement by active and undifferentiated carcinomatous cells. There was fibrosis and an acute inflammatory reaction which was of doubtful protective value. The diagnosis was secondary carcinoma of the lymph nodes.

Case 8.—C. F., a white man, aged 60, married, consulted me on Oct. 15, 1929, for sore throat and swollen glands of the neck. He had always enjoyed good health. On September 1, he had consulted a physician about soreness in the

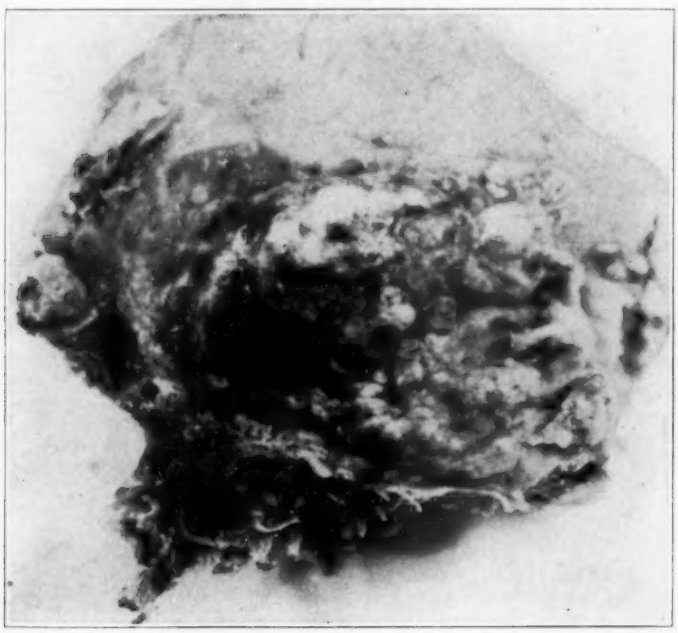

Fig. 17 (case 8).—Growth removed from the posterior border of the tongue.

throat. The condition grew worse, and the glands in the neck on the right side became enlarged; swallowing was painful, and the pain radiated to the right ear and to the right side of head. At times he expectorated some blood.

Laryngeal examination revealed an ulceration on base of the tongue, involving the epiglottis and aryepiglottic fold, and overgrowth of tissue in the glossoepiglottic fold, extending upward to the palatoglossal muscle. Abduction and adduction were normal. Urinalysis, the Wassermann test, blood chemistry and roentgen examination of the chest yielded negative results.

The patient entered the hospital on October 23, and on October 28 a transthyroid pharyngotomy was performed. Under rectal anesthesia, I made a block dissection of the neck, removing the deep cervical fascia and exposing deep structures of the neck. The submaxillary gland was removed; the muscles were separated from the hyoid bone on the right side and the right alae of the thyroid

cartilage on the same side; part of the hyoid bone and thyroid cartilage were then removed. The anterior border of the sternomastoid was sutured to the prevertebral fascia. The lateral wall of the pharynx (fig. 17) was opened and the growth on the posterior border of the tongue inspected. Then the epiglottis, right aryepiglottic fold and posterior third of the tongue as well as part of the palatoglossal muscle were removed by diathermy. The pharynx was closed with two layers of sutures; three drains in the neck and skin were brought together with skin clips. The patient was discharged from the hospital on November 27.

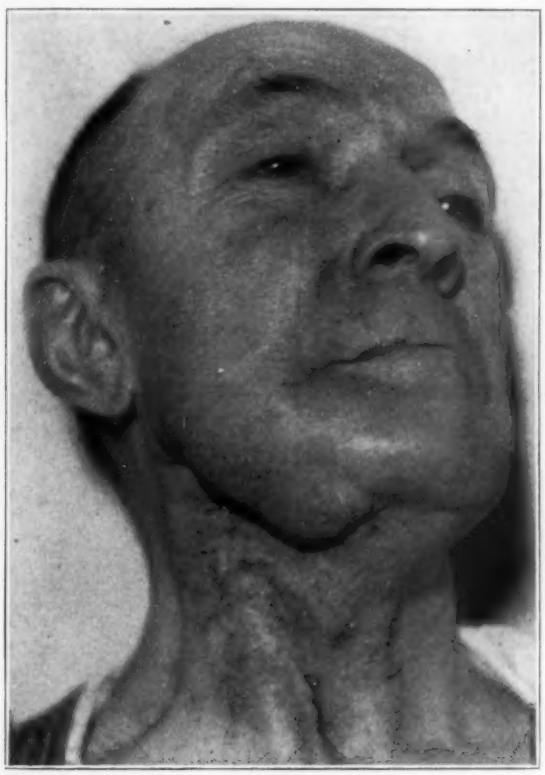

Fig. 18 (case 8).—Appearance of patient after discharge from the hospital.

On pathologic examination the tongue was seen to be ulcerated and had a growth about 2 cm. in diameter at the base. There was an extensive carcinomatous growth of the tongue in which all layers of the surface epithelium were involved. There were numerous epithelial pearls derived from the squamous superficial layer. The growth was extensive and of a high grade of malignancy. Sections from two lymph nodes showed carcinomatous involvement of the same type as the primary growth.

Case 9.—J. K., a white man, aged 43, married, was referred to me on July 3, 1929. The patient had enjoyed good health until nine months (October, 1928) before, when he began to complain of soreness on the right side of the throat and pain radiating to the right ear. Laryngeal examination showed a large ulcera-

tion in the right pyriform sinus involving the lateral wall of the larynx. Urinalysis, the Wassermann test, blood chemistry and roentgen examination of the chest yielded negative results.

Under rectal anesthesia, on July 8, an incision was made on the right side of the neck to form a flap which would cover the defect on the posterior wall of the esophagus, for I intended to remove not only the larynx, but the anterior and posterior wall of the esophagus. The deep fascia and the submaxillary gland were removed. The internal jugular vein was resected on that side. The larynx was skeletonized and the sternomastoid muscle on the right side was sutured to the prevertebral fascia. The trachea was amputated at the second tracheal ring. Then with diathermy the entire mass, including the anterior and posterior wall of the esophagus, was removed. All bleeding was controlled. The upper flap of skin was sutured to the mucous membrane of the anterior portion of the

A B

Fig. 19 (case 9).—*A*, appearance of patient following operation; *B*, appearance of patient on discharge from the hospital.

hypopharynx. The triangular flap of skin was then turned in and sutured to the posterior surface of the hypopharynx and to the skin on the opposite side. The lower portion of this flap was sutured to the posterior wall of the amputated esophagus just above the tracheal opening. This made three sides of a new esophagus. In September, a plastic operation was performed to form the anterior wall. The sides of the trough were undermined and sutured in the median line. This left a raw surface to fill in. A swinging flap was made from the left side of the neck with the pedicle uppermost. The sides were sutured to the undermined edges along the trough. Figure 19 *B* shows the patient at the time of his discharge from the hospital, on Jan. 19, 1930.

Pathologic examination revealed hypertrophy of the epithelium in which the cells showed malignant changes; this was invading the stroma in small groups. The diagnosis was epithelioma.

APPARENT UNILATERAL ABSENCE OF THE SIGMOID SINUS NOTED AT OPERATION *

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In February, 1930, Laff [1] reported a case of unilateral absence of the sigmoid sinus found during dissection. His report included a review of the literature and of the embryologic development of the venous blood channels inside the cranial cavity. He had not found mention of a case in which absence of the sigmoid sinus had been noted at operation. The condition is rarely found in the dissecting room. The case reported herewith was observed at an operation at the Mayo Clinic.

REPORT OF A CASE

A man, aged 31, came to the clinic complaining of earache. He gave a history of having had severe infection of the upper respiratory tract four weeks before coming to the clinic, and during the last week of the infection, the right ear had begun to pain him severely. The day before admission, an attempt at myringotomy had been made, but nothing had been secured. He stated that in childhood he had running ears with tenderness and swelling behind the left ear, and that all symptoms had disappeared after two months. He had had scarlet fever about a year before his present illness.

General examination disclosed that the right tympanic membrane was markedly reddened and bulging, with complete obliteration of all landmarks. There was tenderness over the tip of the right mastoid process. The ears and the nose and throat were otherwise essentially normal. The report of the roentgen examination of the mastoid processes was negative on the left side, and cloudy, graded 2, on the right side. On examination of the roentgenograms, the left mastoid process was seen to be sclerotic, with marked prominence of the groove for the sigmoid sinus. On the right, the mastoid process was diffusely cloudy, with disappearance of the outlines of the air cells. The groove for the sigmoid sinus could not be made out. The Wassermann reaction of the blood was negative. The erythrocytes numbered 4,070,000 and the leukocytes 10,200. The hemoglobin was 70 per cent. A diagnosis was made of acute otitis media, with acute mastoiditis, on the right side.

Myringotomy followed by conservative medical treatment was advised. A large quantity of thick creamy pus was secured under pressure. The patient was hospitalized, and hot compresses were applied to the right mastoid process. The right ear continued to discharge profusely, but the tenderness of the mastoid process was diminished. On the third day after myringotomy, drooping of the posterosuperior wall of the canal was noted for the first time, but tenderness over the

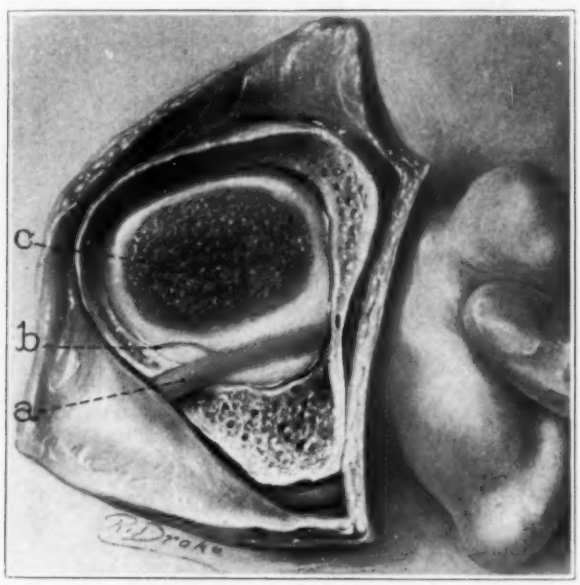
* Submitted for publication, April 16, 1930.

* From the Section on Otolaryngology and Rhinology, the Mayo Clinic.

1. Laff, H. I.: Unilateral Absence of Sigmoid Sinus, Arch. Otolaryng. **11:** 151 (Feb.) 1930.

mastoid process could no longer be elicited. The discharge from the canal of the ear continued to be so profuse that on being wiped out the canal was immediately refilled with the discharge. The drooping of the posterosuperior wall of the canal became more and more pronounced. Although there were no other symptoms, the persistent profuse discharge and the increasing drooping of the posterosuperior wall of the canal were considered sufficient indications for surgical intervention, and mastoidectomy was advised.

When the cortex of the mastoid process was uncovered, the periosteum was found to be adherent, with a well marked groove posteriorly, the petrosquamous suture. The cells of the squamous portion of the mastoid process were small, and the walls of the cells were somewhat sclerotic. A thick bony plate, the petrosquamous lamina, was found above the mastoid antrum, which lay very deep, but the antrum was found to be of approximately normal size. A tract was found



a indicates anomalous superior petrosal sinus; *b*, anomalous lateral sinus; *c*, area of pachymeningitis.

leading upward toward the tegmen mastoideum, which contained many large cells. The dura over the middle fossa was uncovered over an area about 2 cm. in diameter, and a large epidural abscess was found; the dura was covered with thick red granulations. The dura was uncovered over the area of pachymeningitis for an area of about 5 by 4 cm. until normal-appearing tissue was reached in all directions. The tip was then completely removed, and large cells filled with purulent material were found beneath the petrosquamous lamina. These cells were found in the region where the lateral sinus is normally found, and in an attempt to uncover the wall of the sinus, the medial wall of the mastoid process was reached without encountering it. The suspicion then arose that possibly an instance of absence of the sigmoid sinus was present. An attempt, therefore, was made to uncover the sinus at the level of the upper knee. A large vein about two-thirds the size of a normal sigmoid sinus was found, which ran from

just beneath the aditus ad antrum downward and posteriorly. When the wall of the vessel was uncovered, a parasinous abscess was found on the inferior aspect of the sinus.

The lateral sinus was searched for, and nothing could be found but a small vessel about the size of no. 1 catgut, which entered the larger vein at approximately the position which is normally occupied by the upper knee of the sigmoid sinus. During an attempt to uncover the large vein posteriorly, in elevating the periosteum, the vein was torn at the point where it left the bone through a foramen posteriorly occupying the position in which the foramen for the mastoid emissary vein usually lies. The hemorrhage was controlled by a pack, and the vein was packed off where it appeared from beneath the bone anteriorly. The wound was packed with gauze smeared with petrolatum and closed with silk worm sutures, care being taken not to produce pressure against the exposed dura.

The wound was dressed completely in four days; the packs were removed from the superior petrosal sinus without hemorrhage, and the wound was closed. It failed to heal, and a secondary plastic closure was done a month after the first operation. Otherwise, convalescence was uneventful.

It appeared that the large vein found at operation represented the superior petrosal sinus which ran posteriorly and left the mastoid process by the foramen for the emissary vein of the mastoid. The descending portion of the sigmoid sinus was absent, and the lateral sinus was reduced to the size of a thread. An attempt was not made to discover whether a jugular bulb was formed by the inferior petrosal sinus or whether an internal jugular vein was present.

The question arises as to whether the absence of the sigmoid sinus was congenital or possibly due to thrombosis of the sinus during the attack of acute bilateral otitis media which occurred in childhood.

Because of the well developed petrosquamous lamina, with the difference in the size of the air cells in the portion developed from the petrous portion and that from the squamous portion, indicating a tendency toward congenital anomaly, it was thought that the absence of the sigmoid sinus was congenital.

OSTEITIS OF THE PETROUS PYRAMID OF THE TEMPORAL BONE

ASSOCIATED WITH PARALYSIS OF THE EXTERNAL RECTUS *

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AND
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Whatever the cause of these differences, there are wide variations in the number, extent and size of the cells in the temporal bone. As a general rule, the larger the cells the greater is the facility with which infection spreads through them. In the highly pneumatized mastoid processes it is not an unusual operative observation to uncover purulent disease in cells remote from the antrum. It is not clear why infections in the mastoid vary in their predilection for certain pathways. Neumann,[1] however, called attention to this fact.

It is particularly true of the extensively pneumatized, large cell mastoids that the pneumatic structure extends about the bony labyrinthine capsule into the depths of the petrous pyramid. Just as the cells in the mastoid vary in number, extent and size, so do the cells of the petrosa; furthermore, just as in the mastoid an infection at times exercises a predilection for certain pathways, so a similar condition may exist in the pyramid.

In all instances of acute infection of the middle ear associated with involvement of the fifth and sixth nerves due to osteitis that have come under our observation, we have had either operative or roentgen proof of the extensive pneumatization of the mastoid processes. It is conceivable, however, that even when the mastoid process is sclerosed, pneumatic structure may yet exist in the petrosa. A comparison between the space occupied by the sclerosed bony labyrinthine capsule and the perilabyrinthine cells is well illustrated by the accompanying x-ray picture of the petrous pyramid (fig. 18).

As regards the various routes of extension from the middle ear and mastoid cavity to the apex, many paths have been outlined by Perkins,[2]

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* Submitted for publication, April 26, 1930.

* From the Otological and Pathological Departments of Mount Sinai Hospital.

1. Neumann: Die otitische Kleinhirn Abscesse, Vienna, Franz Deuticke, 1907.

2. Perkins, C. E.: Abducens Paralysis and Otitis Media Purulenta, Ann. Otol. **19**:692, 1910.

Chamberlin,[3] Maybaum,[4] Sears,[5] Wheeler,[6] Uffenorde[7] and others in their excellent and comprehensive contributions to the literature on this subject. These pathways are: (1) sublabyrinthine route extending from the tympanum below the labyrinth and internal auditory meatus to the petrous tip; (2) from the mastoid antrum through the subarcuate fossa or the petromastoid canal which passes inward beneath the superior semicircular canal and reaches the layer of cells above the internal auditory meatus to the petrous tip; (3) carotid canal by eroding the bone on the anterior tympanic wall or through one of caroticotympanic foramina which give passage to the carotid branches of the tympanic plexus; (4) layer of cells extending along the eustachian tube from the tympanum to the petrous tip; (5) along the superior or inferior petrosal sinus to the apex; (6) extension of an extradural abscess of petrosa to the apex; (7) indirect trauma at operation or otherwise.

It is maintained that the list is not complete owing to the marked variability in the anatomy of the temporal bone. However, the intensity or the severity of the osteitis may vary, and the extension of the infectious process may take place through one or several of the pathways described.

It cannot be denied, however, that the majority of infections of the petrous pyramid that reach the apex do so by direct extension through the cells.

All infections in the petrous pyramid do not necessarily extend to the apex. Nor do they invariably involve the sixth nerve, even though the apex is the site of diffuse inflammatory changes or an abscess formation. This was pointed out by Lange in 1909, by Wagener[8] in 1911 and by Perkins,[2] who stated that he encountered epidural abscesses in the region of the apex without any apparent effect on the sixth nerve. Obviously, then, in the face of this possibility, the extent of the inflammatory process in the bone toward the apex cannot be

3. Chamberlin, W. B.: Gradenigo Symptom Complex, Atlantic M. J. **27**:566, 1924.

4. Maybaum, J. L.: Two Cases of Gardenigo Syndrome? Laryngoscope **30**: 138 (March) 1920.

5. Sears, W. H.: Isolated Paralysis of External Rectus in Acute Suppurative Otitis Media, Pennsylvania M. J. **13**:844 (Aug.) 1910; Gradenigo Syndrome, Laryngoscope **37**:33, 1927.

6. Wheeler, J. M.: Paralysis of Sixth Cranial Nerve Associated with Otitis Media, Tr. Sect. Ophth., A. M. A., 1918, p. 51.

7. Uffenorde: Zur Klinik der Falle von Mittelohr-Eiterung mit tiefen perilabyrintharen Herden, Arch. f. Ohrenh. **105**:87, 1919; Eine zu vielfachen Verwicklungen führende sehr ausgedehnte perilabyrinthare Eiterung im histologischen Bilder, ibid. **122**:288, 1929.

8. Lange and Wagener, quoted by Vogel, H.: Der sogenannte Gradenigosche Symptom Komplex, Internat. Zentralbl. f. Ohrenh. **19**:1, 1922.

gaged by the presence or absence of involvement of the fifth or sixth nerve. Moreover, an infection in the pyramid may perforate the bony cortex before it reaches the apex. At the site of such a perforation, there may be an extradural infection, which may either remain localized or extend mesially along the dura and involve the fifth and sixth nerves separately or together. Such a perforation, with its attendant infection of the dura, may result in meningitis, local or diffuse, intradural abscess or a localized intramedullary process, i. e., abscess of the temporal lobe or cerebellar abscess.

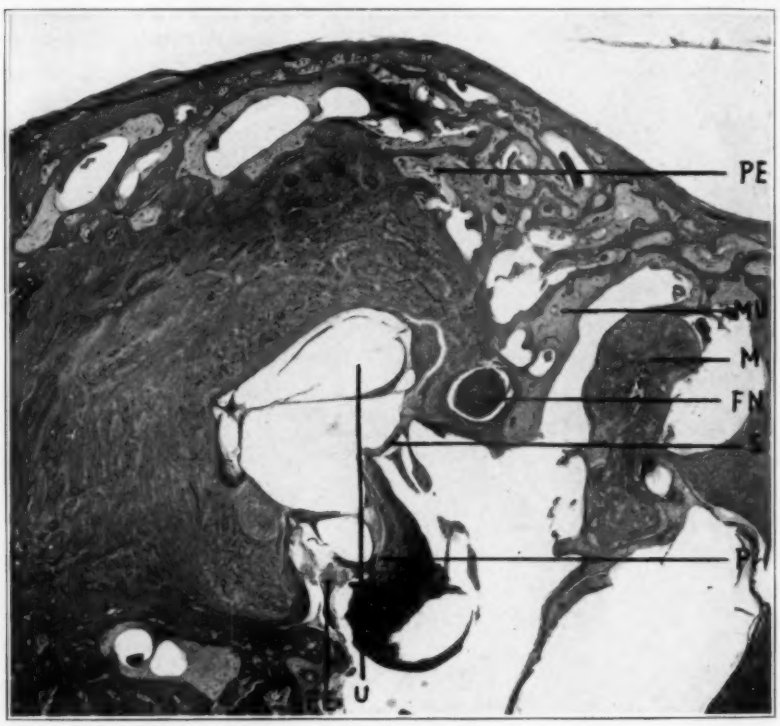

Fig. 1.—Vertical section through the tympanum and vestibule, showing purulent exudate in the tympanic cavity, thickened submucosa with the spread of the infectious process to the adjacent pneumatic cells near the middle fossa and extending posteriorly almost up to the posterior fossa. *FN* indicates the facial nerve; *M*, the malleus; *MU*, thickened mucosa; *Pr*, promontory; *PE*, purulent exudate in the tympanum and adjacent cells; *S*, stapes, and *U*, utricle.

In its pathway through the petrosa, involving the fifth and sixth nerves, the infection occasionally also invades either the facial nerve or the internal ear. Cases of osteitis with involvement of the fifth and sixth nerves, which we have examined histologically and which are presented here, have shown neither the facial nerve nor the labyrinth

affected by the disease. Cases have been reported, however, with concomitant involvement of either the facial nerve or the labyrinth or both, and we have seen several such instances clinically.

There is still some question as to the exact nature of the pathologic process that occurs when the infection reaches the apex after having traveled through the pyramid. The fifth nerve is undoubtedly involved at its ganglion; the sixth nerve, in all probability, in its course through Dorello's canal. Our sections show definitely a periganglionitis and a

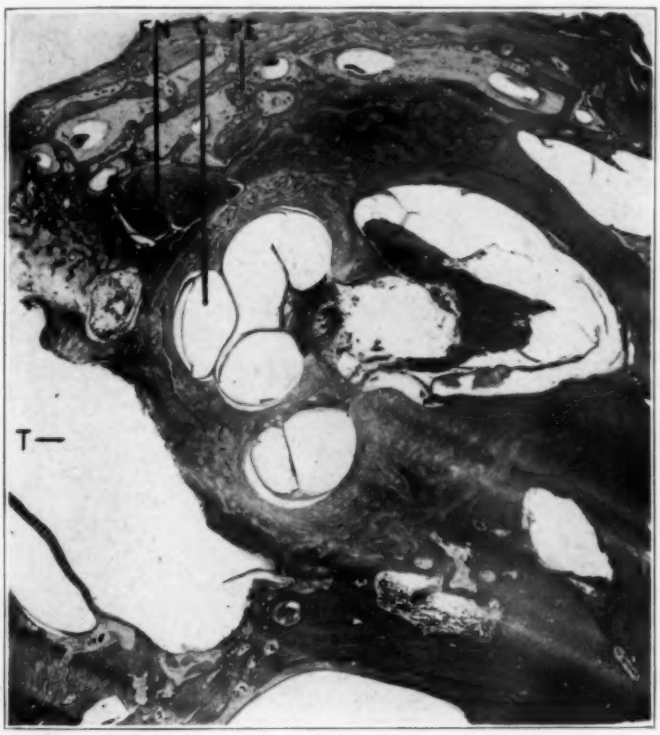

Fig. 2.—Section through the cochlea, showing the spread of the infection medially in the pneumatic cells beneath the floor of the middle fossa. Within these pneumatic cells granulation tissue, purulent exudate and new bone formation may be discerned. *C* indicates the cochlea; *FN*, the facial nerve; *PE*, purulent exudate, and *T*, tympanum.

ganglionitis. Whether in the cases in which recovery takes place the process is solely a pachymeningitis or whether there is a coexistent localized leptomeningitis is still problematic. Either or both could be responsible for the paralysis of the sixth nerve. In all probability, there are wide variations in the intensity and the extent of inflammatory changes in the petrosa following infection. It seems, however, that in

those cases in which recovery occurs spontaneously, the inflammation is much more likely to be mild and confined to the bone. In the latter case, there is surely a greater facility for drainage and spontaneous resolution than there could be in those instances of severe extensive infections which, having perforated the bony cortex, involve the intracranial contents.

Among other theories that have been advanced to explain particularly the involvement of the sixth nerve are: (1) its involvement as a labyrinthine reflex; (2) involvement caused by toxic influenza, rheumatic fever, diphtheria, etc. With regard to the toxic influence of influenza, in the cases reported in which this theory has been held there has usually

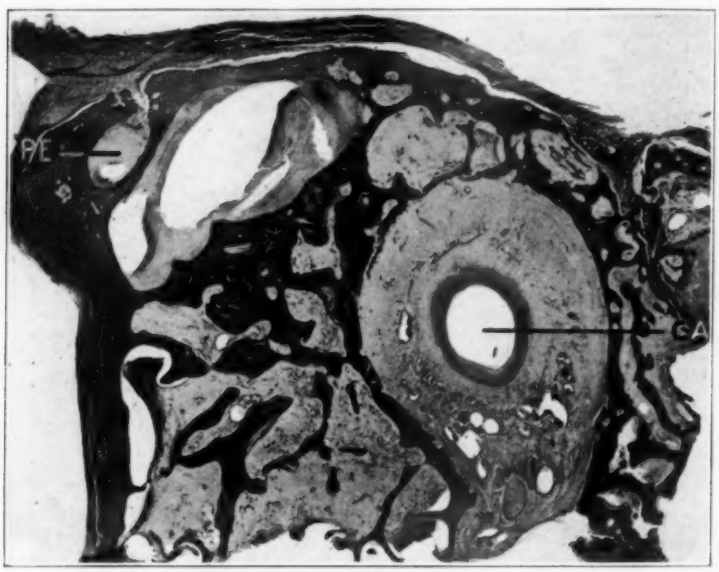

Fig. 3.—Section of the petrous pyramid medial to the tip of the cochlea, showing the infection in the large pneumatic cells near the superior posterior margin and the communication of one of these cells with the dura. *CA* indicates the carotid artery; *PE*, the purulent exudate.

been a concomitant otitis with or without mastoid involvement. One patient with a unilateral otitis had a bilateral paralysis of the sixth nerve. One of the patients in our series presented no clinical evidences of mastoiditis with a resolved middle ear, and recovered completely without any operative intervention. Nevertheless, this patient probably also had an inflammatory process in the pyramid which healed spontaneously.

It is our opinion that in the cases that we studied the paralysis of the abducens nerve and the involvement of the gasserian ganglion were early manifestations of a pachymeningitis which in all probability was at first circumscribed and later terminated in a diffuse purulent

leptomeningitis. It is during the stage of localized meningitis at the apex with the associated edema, swelling and pressure that the sixth nerve is involved. It is at this site that the nerve and its vessels pass through Dorello's canal, which is bridged across by the petrosphenoidal ligament. This view is in accordance with that of many American and European authors. In a group of temporal bones removed at autopsy

Fig. 4.—Same section as in figure 3, under higher magnification. *D* indicates the dura; *PE*, the purulent exudate in communication with the dura.

we have traced the sixth nerve through Dorello's canal, and have found that the petrosphenoidal ligament varies in size and shape (figs. 12 and 18), as has been pointed out (Vail [9]).

The causative factors in the production of paralysis of the abducens nerve and its method of extension have thus been demonstrated histo-

9. Vail, H. H.: Anatomical Studies of Dorello's Canal, Laryngoscope **32:** 569 (Aug.) 1922.

logically in three cases. That there are causes other than osteitis of the petrous pyramid is unquestionable. Perkins,[2] who made a study of 95 cases, ascertained the cause with reasonable assurance in 33; namely, sinus thrombosis, 2; meningitis, 3 (unknown route of infection); labyrinth disease, 4; abscess in posterior fossa, 9; abscess in middle fossa, 2; osteitis or disease of petrous apex, 13.

Ever since Gradenigo [10] published his first monograph in 1904 on the syndrome bearing his name—suppuration of the middle ear, pain in the temporoparietal region and paralysis of the abducens nerve—considerable interest regarding its pathogenesis has been stimulated both

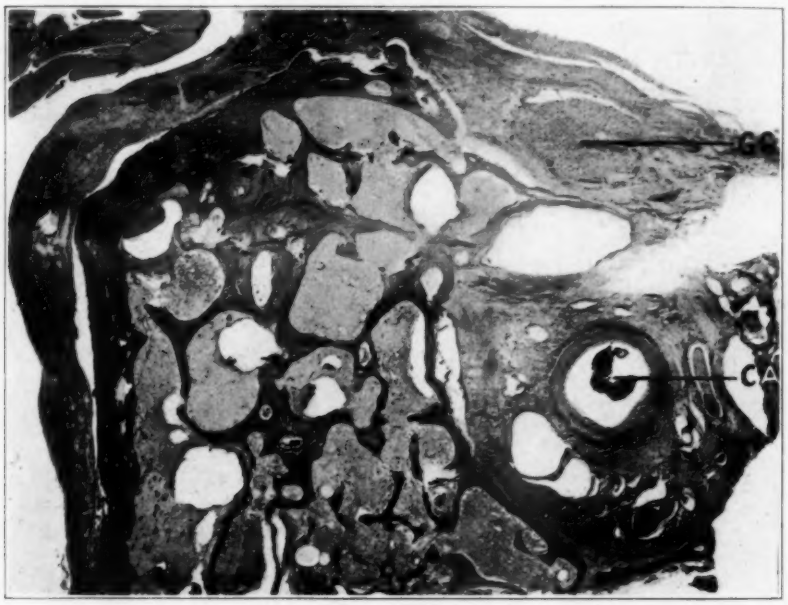

Fig. 5.—Section through the gasserian ganglion. The dura about the ganglion is markedly thickened and edematous, being only slightly infiltrated. *CA* indicates the carotid artery; *GG*, the gasserian ganglion, and *VN*, the fifth nerve.

in this country and abroad. Numerous contributions to the literature on the subject have since been made; various theories have been expounded and explanations offered as to the underlying pathologic changes involving the fifth and sixth nerves. The various explanations, briefly, are: circumscribed suppurative leptomeningitis, localized pachymeningitis, serous meningitis, inflammatory edema of the tip of the petrous pyramid, toxic neuritis and reflex (labyrinthine).

10. Gradenigo: Ueber circumscripte Leptomeningitis mit spinalen Symptomen und über Paralyse des Nervus abducens otitischen Ursprungs, Arch. f. Ohrenh. **62**:255, 1904.

The amount of pathologic data, both macroscopic and particularly microscopic, offered in the literature is surprisingly meager. We present here a study, both grossly and microscopically, of the temporal bones in three cases of otitic suppuration associated with paralysis of the external rectus muscle, with the object of ascertaining, if possible, a causal relationship.

The temporal bones have been studied and each has been sectioned serially from the mastoid process to the apex of the petrous pyramid, so that, if possible, the infectious process could be traced throughout. This process could be demonstrated in three of the four cases; in the fourth, the cause of the external rectus palsy could not be determined,

Fig. 6.—Section through the tympanum in the region of the promontory, showing the severe inflammatory process in the pneumatic cells in the region of the superior posterior margin of the petrous pyramid with formation of Howship's lacunae lined by osteoclasts and beginning bone sequestration. Secondary involvement of the lining dura with the production of an extradural abscess is shown. *A* indicates the extradural abscess; *Pr*, the promontory; *S*, the sequestrum, and *T*, the tympanum.

and it is not included. In the first case, the infection spread from the tympanum and antrum to the adjacent cells in the epitympanic roof and from there by direct contiguity through the cells beneath the floor of the middle fossa and superficial to the posterior margin of the superior semicircular canal but not involving the latter, then mesially toward the apex of the petrous pyramid. The infection can be seen to involve the

dura of the middle fossa at about the depth of the internal auditory meatus.

In the second case the infection spread also from the tympanum and antrum by direct extension through the adjacent cells of the epitympanum, but here the break into the dura occurred in the posterior fossa.

Fig. 7.—Same section as in figure 6, under higher magnification. Lacunae formation, giant cells and beginning bony sequestration may be seen. Severe secondary inflammation of the adjacent dura and a subdural abscess are also demonstrable. *A* indicates the extradural abscess; *D*, the inflamed thickened dura, and *S*, the sequestrum.

In the third case the infection spread from the tympanum and eustachian tube through bony dehiscences (caroticotympanica foramina) into the carotid canal and then up to the apex of the petrous pyramid.

It is perhaps advisable at this time to summarize briefly the history and the pathologic observations in each case.

CASES STUDIED GROSSLY AND MICROSCOPICALLY

CASE 1.—*History.*—S. L., a woman, aged 32, was admitted to the service of Dr. Elsberg on May 8, 1928, with a history of grip two months prior to admission followed by acute otitis media of the left side, for which a myringotomy was performed. Discharge continued from the ear for two days, followed by postauricular pain and mastoid tenderness. A simple mastoidectomy had been performed at another hospital. Following the operation the patient complained of severe

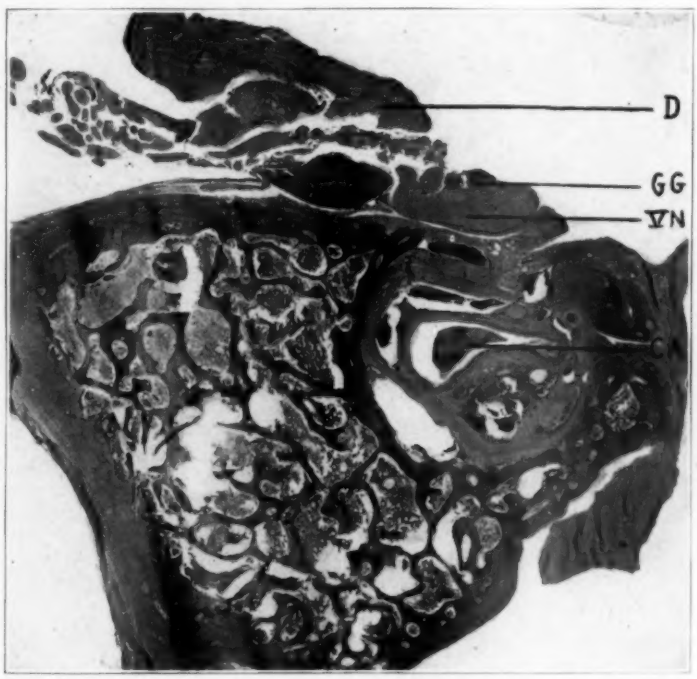

Fig. 8.—Section through the gasserian ganglion, showing inflammatory thickening of the dura about the ganglion and the fifth nerve. *CA* indicates the carotid artery; *D*, the dura; *GG*, the gasserian ganglion, and *VN*, the fifth nerve.

temporoparietal headache necessitating the use of narcotics. Five days prior to admission, chills, a rise in temperature to 103.6 F. and projectile vomiting occurred.

Physical Examination.—On admission the temperature was 101 F. There were left motor and sensory paralysis of the fifth nerve and paralysis of the left sixth and the left peripheral seventh nerves. The following day spasticity of the neck was present. Spinal puncture revealed cloudy fluid, 600 cells and no organisms on smear or culture. The blood culture was negative. The mastoid wound was granulating; the tympanic membrane was thickened, and there was no discharge. Hearing was impaired. In view of the physical signs, the negative blood culture and the comparatively low temperature, the diagnosis rested between abscess of

the brain and osteitis of the petrous pyramid. Exploratory craniotomy for abscess of the brain was performed with negative observations. Following the operation, the patient made a decided turn for the worse; the meningeal signs increased, the cell count increased and *Streptococcus hemolyticus* was recovered from the fluid. The patient died fifteen days after operation.

Postmortem Examination.—On postmortem examination, a purulent meningitis was found. There were no gross pathologic changes in the petrous pyramid.

Histologic examination of the temporal bone revealed: The pneumatic cells about the mastoid antrum, and particularly in the region of the epitympanum, showed evidence of an acute inflammatory process. The cavities were filled with

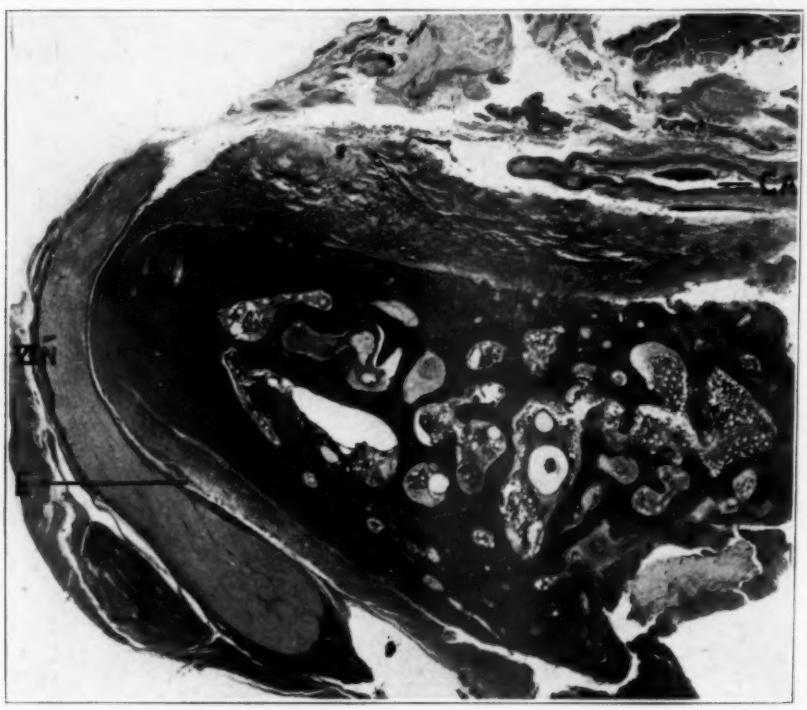

Fig. 9.—Section through the apex, showing very slight exudate about the sixth nerve. *VI N* indicates the sixth nerve; *CA*, the carotid artery, and *E*, exudate.

purulent exudate consisting of polymorphonuclear leukocytes and lymphocytes. The mucosal lining was thickened and infiltrated with cellular elements. This inflammatory process could be traced from the epitympanic cells to the adjacent cells along the floor of the middle fossa (fig. 1) superficial to the superior semicircular canal but not involving the bony capsule extending posteriorly almost to the posterior fossa and below the horizontal portion of the facial nerve. The bony septums were more or less intact. The inflammatory changes were more severe in this region than in the antral region. The various stages of the inflammatory process could be seen here with evidence of repair, i.e., new bone formation. As the cochlea was approached, the changes in the cells superior to the bony labyrinth were even more marked (fig. 2). The pneumatic cells were

definitely increased in size at this location (usually larger normally) and became larger as the apex of the petrous pyramid was approached. At about the depth of the internal auditory meatus in the region of the angle between the superior and posterior surfaces of the petrous pyramids near the superior petrosal sinus, the bony cortex had undergone necrosis and the overlying dura had become secondarily involved, resulting in meningitis (figs. 3 and 4). At this site the dura was considerably thickened and markedly infiltrated with cellular elements. The inner layers of the dura "kambium" layer were the more involved. The dura in the region about the gasserian ganglion and at the tip showed only slight changes; that is, it was thickened, but was only slightly infiltrated with cells (fig. 5).

Comment.—In this case the progress of the symptoms and pathologic observations can readily be correlated. The patient evidently had a virulent infection of the middle ear which undoubtedly involved the

Fig. 10.—Section through the tympanum and vestibule. Purulent exudate may be seen in the tympanic cavity. *E* indicates exudate in the tympanum; *FN*, the facial nerve; *S*, the stapes, and *U*, the utricles.

mastoid cells simultaneously or soon afterward and for which an exenteration was performed. Following the operation, the patient still complained of temporoparietal headache. This, in all probability, was due to the secondary involvement of the dura of the middle fossa and of the gasserian ganglion. The headache subsided with the arrest of the meningeal irritation. The infectious process, however, continued by direct extension through the cells of the petrous pyramid and finally broke through the cortex of the latter near the superior posterior margin at about the depth of the internal auditory meatus. The infection then spread along the dura, involving the gasserian ganglion and the sixth and seventh nerves. This process of infection through the bone was

associated with very few symptoms, as in the interim the patient was free from pain until the meninges were further involved nearer the apex. It was at this time (five days prior to admission) that the meningeal signs appeared—projectile vomiting, hyperpyrexia and involvement of the fifth, sixth and seventh nerves. The spinal fluid at first was free from organisms. Following the cranial exploration, bacterial invasion into the spinal canal was manifest. It is also of interest to note that the infection, as it advanced toward the apex, was more severe in its course, although it was apparently subsiding behind. On admission, the middle ear had practically resolved.

Case 2.—*History.*—L. S., a girl, aged 5, was admitted to the pediatric service of Dr. Schick on July 3, 1928, with a history of chronic discharging ears since the age of 1½ years. About two months previously, a mastoidectomy was performed at another hospital, with apparently good recovery. Two weeks prior to admis-

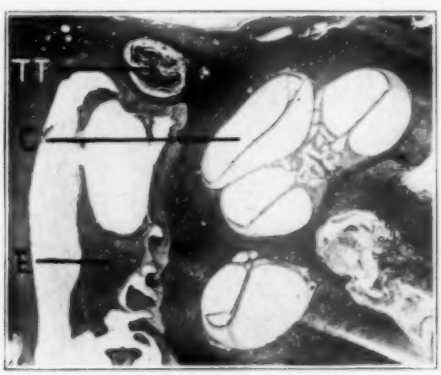

Fig. 11.—Section through the cochlea and eustachian tube, showing the inflamatory process lining the eustachian cavity and purulent exudate within. *C* indicates the cochlea; *E*, the exudate in the eustachian tube, and *TT*, the tensor tympani muscle.

sion, headache, vomiting and a rise in temperature of from 102 to 104 F. occurred; there were convulsions on the day of admission. No discharge from the ears was noted.

Examination.—The right ear was normal. There was a scant discharge from the mastoid wound of the left ear; the drum was thickened, but landmarks were visible. The temperature was 104 F. Left abducens paralysis, stiff neck and bilateral Kernig's signs were present. Lumbar puncture showed turbid fluid; streptococci were recovered on smear and culture. The patient died two days after admission.

Postmortem Examination.—Purulent meningitis was present particularly at the surface of the brain. In the region of the internal auditory meatus, somewhat superiorly and posteriorly, the dura appeared thickened and bulged for an area about 1 cm. in diameter. On palpation, the presence of fluid between the dura and the underlying bone could be discerned. The diagnosis of a deep subdural abscess was made. There were no gross changes at the apex of the petrous pyramid.

Histologic examination of the temporal bone showed: inflammation of the pneumatic cells about the antrum, particularly in the epitympanum, purulent exudate in the cells and marked thickening of the mucosal lining. There was considerable submucosal infiltration; granulation tissue was found, with almost complete obliteration of some of the cells. This inflammatory process extended by direct contiguity through the cells as far as the bony labyrinth below and almost to the posterior margin of the pyramid. At the depth of the internal auditory meatus there was marked necrosis of bone with lacunae lined by many osteoclasts. Here the bone had been eroded at the posterior surface, and the dura was undermined with the formation of an extradural abscess (figs. 6 and 7). It was this abscess that was observed grossly on postmortem examination. The dura was separated from the bone, was considerably thickened and showed the presence of

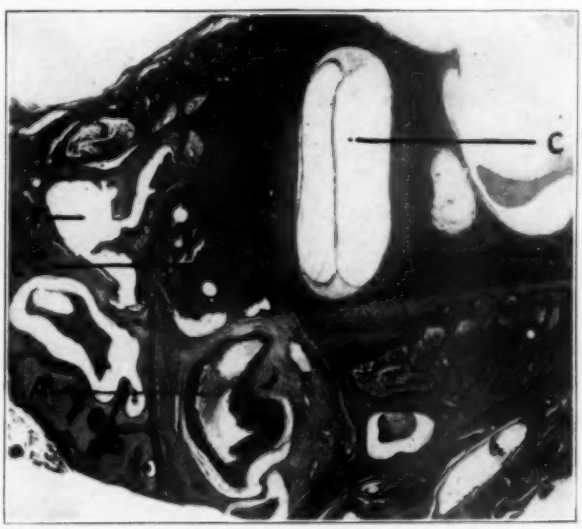

Fig. 12.—Somewhat more anterior to figure 10, showing communication between the eustachian cavity and the carotid canal by means of a dehiscence. Purulent exudate may be seen in both the eustachian tube and the carotid canal. *C* indicates the cochlea; *CA*, the carotid artery; *CC*, the carotid canal with pus within, and *ET*, the enstachian tube.

large numbers of cells and granulation tissue. The inflammatory changes were less marked at the apex (figs. 8 and 9).

Comment.—The extension of the infectious process in this case is similar to that in the first—by direct contiguity through the cells of the petrous pyramid. Here, however, the cortex of the posterior fossa was eroded, with the production of a deep subdural abscess and a secondary bacterial meningitis with involvement of the sixth nerve. In this case, as in the first, the middle ear and the mastoid cavity showed evidences of resolution, while the infection progressed within the substance of the petrous pyramid. Also this case resembles somewhat that of Lombard,

described by Gradenigo [11] in his second publication in which at autopsy a deep subdural abscess extending from the internal auditory meatus to the posterior clinoid process was found. The gasserian ganglion and the sixth nerve lay free in the pus.

Case 3.—*History.*—A. H., a boy, aged 4, was admitted to the pediatric service of Dr. Schick at Mount Sinai Hospital on April 24, 1928, with a history of otalgia of the right side twelve days prior to admission, associated with a rise in temperature. One week later, because of a rise in temperature to 105 F., myringotomy was performed. The following day paralysis of the external rectus muscle developed on the right side; stiff neck and vomiting were present. Three days later there was a bilateral Kernig sign.

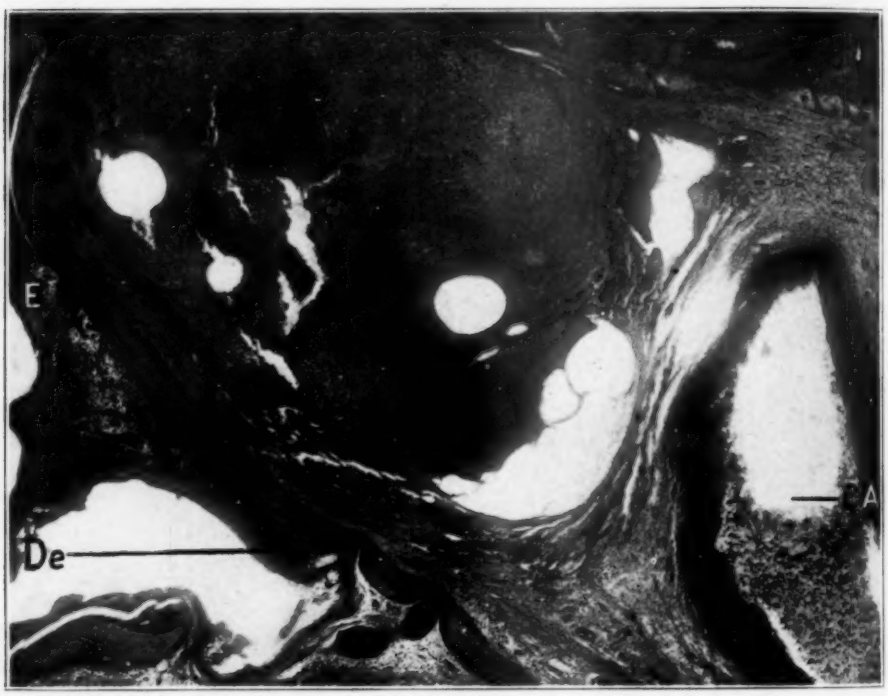

Fig. 13.—Same specimen as in figure 12, under higher magnification. *CA* indicates the carotid artery; *De*, the dehiscence between the carotid canal and the eustachian tube, and *E*, purulent exudate.

Examination.—Locally, there was a thin purulent discharge from the myringotomy wound. No other local signs were obtained. Spinal puncture revealed cloudy fluid under slightly increased pressure. The blood showed: 1,500 cells with 75 per cent polymorphonuclears. Pneumococcus III was recovered on culture. A blood culture was negative. The patient had a rapid downhill course, and died two days later.

11. Gradenigo: Ueber die Paralyse des Nervus abducens bei Otitis, Arch. f. Ohrenh. **74**:149, 1907.

Postmortem Examination.—A purulent bacterial meningitis was present. There were no gross abnormalities of the temporal bone.

Histologic examination of the temporal bone revealed purulent exudate in the tympanum (fig. 10) and the eustachian tube (fig. 11), consisting of polymorphonuclear leukocytes and lymphocytes. The mucous membrane lining the tympanum and eustachian tube was intact almost throughout its entirety, except for perforation in the membrana tympani. There was marked submucosal thickening with lymphocytic infiltration. In the region of the knee of the carotid canal there was a large accumulation of pus, giving the appearance of a localized abscess. This pus cavity communicated directly with the eustachian tube through bony dehiscences (figs. 12 nd 13). The purulent exudate could be traced to the adjacent cells of the petrous pyramid, and its destructive effect on the bony septums could be seen by the production of large Howship's lacunae lined by many osteoclasts. This

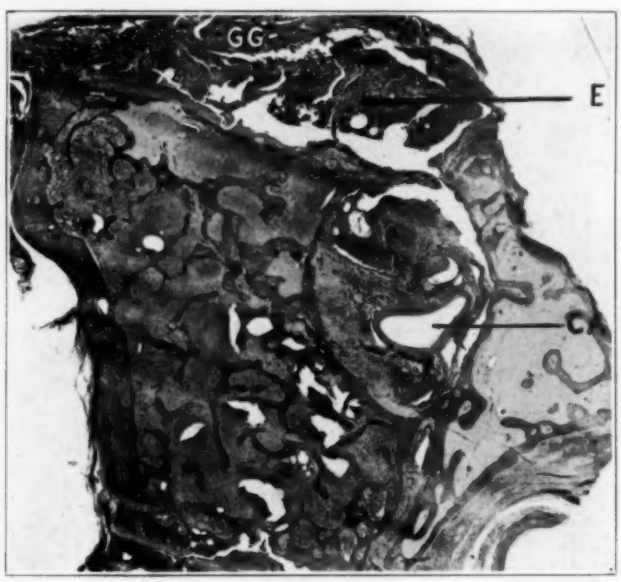

Fig. 14.—Section through the gasserian ganglion, showing marked purulent exudate about the ganglion and in the carotid canal. *CA* indicates the carotid artery; *GG*, the gasserian ganglion, and *E*, the purulent exudate about the gasserian ganglion.

process extended up to the apex of the petrous pyramid. In the region of the gasserian ganglion, the infection produced necrosis of the bony cortex, perforating it, with resulting extravasation of pus around the gasserian ganglion (fig. 14) and the sixth nerve (fig. 15).

Comment.—This case differs from the others in that the infection spread from the eustachian tube into the carotid canal through bony dehiscences. The process then extended along the canal, invading the adjacent bone cells of the petrous pyramid and finally involving the dura in the region of the gasserian ganglion. These dehiscences of the

foramina are found not too infrequently, particularly in young patients, and at times serve for the passage of the carotid branches of the tympanic plexus. Shambaugh,[12] in a lecture, pointed out these dehiscences between the middle ear cavity and the carotid canal. Also, Urbantchich and Bezold demonstrated an opening in the superior posterior part of the ostium of the eustachian tube somewhat posterior to the promontory. In some cases a probe could be passed through this opening up to the tip of the petrous pyramid.

Fig. 15.—Section through the apex, showing purulent exudate in the vicinity of the sixth nerve. *VI N* indicates the sixth nerve; *CA*, the carotid artery, and *PE*, purulent exudate.

GENERAL COMMENT

These cases, as may be gathered from the histories, cannot be classified as those representing the Gradenigo syndrome in the strict sense of the term as originally used, but would rather coincide with Gradenigo's third group in which the cases terminate fatally, with diffuse meningitis.

12. Shambaugh, quoted by Sears (footnote 5).

A review of the literature regarding the association of suppuration of the ear with involvement of the fifth and sixth nerves gives evidence of differences of opinion as to the underlying pathologic basis. On the one hand, a series of authors quoted by Vogel,[13] i. e., Rimini (1906), Grivot (1908), Hedon (1910), Blanc (1910), Malan (1911), Kerandren (1912), Pritchard (1913), Lergard (1918) and Chevanne (1920), expressed the belief that the etiology of the lesion of the nerve is an osteitis of the petrous pyramid. On the other hand, an equally large series, quoted by the same author, i. e., Kepp (1879), Jansen (1893), Jurgenmeyer (1898), Habermann (1898), Kollner (1908), Korner (1908), Luc (1908), Mariefka (1909) and Sterling (1910) ascribed the condition to diffuse meningitis, so that Vogel would discard the term Gradenigo syndrome entirely.

It must be obvious that no single constant etiologic factor exists which is responsible for that triad of phenomena that Gradenigo

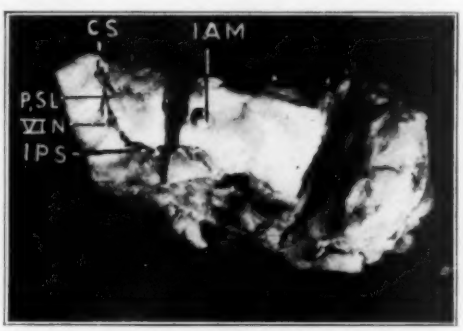

Fig. 16.—Gross specimen of the temporal bone (normal) dissected to show Dorello's canal, petrosphenoidal ligament and course of the sixth nerve. *CS* indicates the cavernous sinus; *IAM*, the internal auditory meatus; *IPS*, the inferior petrosal sinus, and *VI N*, the sixth nerve.

described, even though infection of a petrous pyramid may exist in the majority of instances. Yet even in the presence of generalized meningitis we have found evidence of infection of the petrous pyramid, so that either the meningitis or the disease of the bone might have been responsible for the involvement of the fifth and sixth nerves. Naturally, when the fifth and sixth nerves are affected during or after the appearance of meningeal signs, along with other nerves remote from the pyramid, disease in the latter cannot be regarded as the direct cause of the involvement of the cranial nerve.

With regard to the symptoms of the so-called Gradenigo syndrome, the subjective and objective phenomena are so well known that they

13. Vogel, H.: Der sogenannte Gradenigosche Symptom Komplex, Internat. Zentralbl. f. Ohrenh. **19**:1, 1922.

require but a brief description. The triad of inflammation of the middle ear, with or without mastoiditis, the pain, frequently paroxysmal, usually referred to the temporal region or to the eye, and palsy of the external rectus muscle are the factors grouped as the syndrome. The pain and the paralysis may occur while the middle ear is discharging or at any stage up to or after complete resolution. This fact, which has been proved clinically, is supported also by histologic proof.

Fig. 17.—Section through the apex of the petrous pyramid (normal), showing the course of the sixth nerve before it enters the temporal bone. *VI N* indicates the sixth nerve; *PSL*, the petrosphenoidal ligament, and *CA*, the carotid artery.

The pain may be severe or slight, or it may be absent. The onset of paralysis of the sixth nerve may occur at any time following the infection of the middle ear, within from a week to twelve weeks. Both the temperature and the changes in the white blood count vary with the nature of the pathologic process.

The prognosis in suppuration of the middle ear with or without mastoid involvement complicated by a paralysis of the abducens nerve is comparatively favorable, Gradenigo reported four deaths in fifty-

seven cases, a mortality of 8 per cent; Perkins, eleven deaths in ninety-five cases, a mortality of 11 per cent, and Baldinweck (quoted by Schneideman in the discussion of Sear's paper), eleven deaths in sixty-seven cases, a mortality of 16 per cent. There is no doubt that if signs of mastoiditis appear in the course of an involvement of the middle ear complicated by paralysis of the abducens nerve, a thorough and complete mastoidectomy is the proper procedure. It is advisable to keep the wound open for a considerable length of time. This is particularly true in cases of pneumococcus III infection. But in the event that the paralysis follows an exenteration of the mastoid, as is frequently the case, what should be the line of treatment? Since the prognosis in these cases is comparatively good, as was stated, since a correct diagnosis of suppuration in the petrous pyramid is uncertain and

Fig. 18.—Roentgenogram of the petrous pyramid.

since the accessibility to this region is rather difficult, conservative measures are most frequently indicated. It has been our experience that those instances in which the pain ceases with the onset of paralysis of the sixth nerve offer a better prognosis than those in which the pain persists. The cases in which pain persists or in which pain after having disappeared recurs are more liable to progress to intracranial complications. So, too, paralysis of the sixth nerve may disappear, only to recur with the development of meningitis. To illustrate the indications for conservative treatment, the cases of two patients who were admitted to the otologic service at Mount Sinai Hospital are cited briefly.

M. L. (Case 7 under "Report of Cases"), a boy, aged 8, had a six weeks' history of acute otitis media when simple mastoidectomy was performed. Two days after operation external rectus palsy developed, followed by a vertical nystagmus upward on the following day and pain in the teeth three days later. Twenty-five

days after the onset of the palsy of the external rectus muscle, it had completely returned to normal, and the child made a good recovery.

In S. M. (Case 8 under "Report of Cases"), a boy, aged 12, following mastoidectomy, i.e., twenty-five days after the onset of the acute otitis, paralysis of the abducens nerve developed without any pain or headache. The paralysis showed signs of improvement fourteen days later, and cleared up one week following, that is, twenty-one days after its onset. At about this time, however, the patient vomited and had a rise in temperature to 103 F. and a ptosis of the upper lid, no other localizing meningeal signs being present. Two days later, the patient showed definite signs of improvement, and he went on to a complete recovery.

If, however, after complete mastoidectomy progressive signs of a meningeal involvement appear, as evidenced by the physical signs and examination of the cerebrospinal fluid, when the prognosis for recovery is questionable further exploration is definitely indicated, namely, an approach to the apex of the petrous pyramid. This operation is not beyond surgical means, since it was performed by Gorris (described by Streit [14]) many years ago, and more recently by Monroe,[15] Eagleton, Bowers and Richards [16] and others. Otto Mayer [17] described an interesting case in the September, 1929, issue of the *Monatsschrift für Ohrenheilkunde und Laryngo-Rhinologie,* in which he reached the apex of the petrous pyramid through a group of cells below the promontory and found an abscess, which he drained. The patient made a complete recovery.

No definite outline as to the method of approach can be formulated, since one must be guided by the symptoms and pathologic observations at operation; a supralabyrinthine, a postlabyrinthine or a sublabyrinthine route may be the one of choice depending on the lead gained. It is assumed that the labyrinth is intact. In the event that during this exploration a deep subdural abscess on the superior or posterior surface of the petrous pyramid is encountered, its drainage may be all that is required.

It is fully understood that paralysis of the abducens nerve may not be due to an osteitis of the petrous pyramid, and a correct diagnosis cannot always be made; nevertheless, it is our opinion that in those cases in which the prognosis is not favorable, namely, on the appearance of progressive meningeal signs, this method of approach is the most rational, and it is hoped that a number of patients who otherwise would die will be helped by this procedure.

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14. Streit: Arch. f. Ohrenh. **59**:169, 1903.

15. Monroe, quoted by Ulrich, K.: Zur lehre von des otogenen Abducenlahmung, Ztschr. f. Hals-, Nasen- u. Ohrenh. **9**:403, 1924.

16. Eagleton; Bowers and Richards: Personal communication to the authors.

17. Mayer, Otto: Gradenogscher Symptom Komplex bei akuter Mittelohreiterung, Monatschr. f. Ohrenh. **63**:970, 1929.

CONCLUSIONS

1. Osteitis of the petrous bone as a causative factor in the production of paralysis of the abducens nerve is much more prevalent than is believed. This may not always be demonstrable macroscopically, but may be ascertained by histologic study.

2. The diagnosis of osteitis of the apex of the petrous pyramid can be made correctly in many cases.

3. The infection may subside in the tympanum and mastoid cavity but may progress toward the apex of the petrous pyramid with increasing severity.

4. The infection may extend through the bony substance of the petrous pyramid and reach the apex, or may perforate the cortex and involve the dura at any point in its course and then spread to the apex.

5. Anatomic variations of the petrous pyramid play an important rôle in the spread of infection in that structure. In the cases in which recovery occurs spontaneously, the infection probably is confined to the bone.

6. As there is no single anatomicopathologic basis for the so-called Gradenigo syndrome, the value of the term is doubtful.

7. Surgical approach to the apex of the petrous pyramid is indicated in the presence of progressive meningeal signs.

REPORT OF CASES

The following are brief clinical reports of twelve cases that have come under our personal observation. Of the twelve patients, ten recovered and two died.

CASE 4.—H. R., aged 6, was admitted to Mount Sinai Hospital on Jan. 4, 1928. At the time of the mastoid operation, done on the day of admission, the patient had the physical evidence of a surgical mastoiditis. X-ray films showed a pneumatized mastoid. Following the operation, the temperature was always as high as 100.5 F. One week after discharge from the hospital, on January 17, the patient complained of pain in the left eye and along the lower left jaw. One week after the onset of the pain, paralysis of the sixth nerve developed. Following the onset of the disease of the sixth nerve, there was very little pain. The nerve gradually came back to normal, and the patient made an uneventful recovery.

The hemolytic streptococcus was recovered.

CASE 5.—J. G., aged 3½, was admitted to the hospital on June 22, 1929. One month before admission, paralysis of the sixth nerve developed, associated with suppuration in the middle ear, which lasted two and one-half weeks and then disappeared. Not until admission to the hospital was the evidence of mastoid disease present, at which time a simple mastoid operation was done. The patient had a perisinus abscess. He was discharged on July 2.

The hemolytic streptococcus was recovered.

CASE 6.—H. H., aged 17, was admitted to the hospital on June 3. The patient was operated on for mastoiditis before being seen by us. Eight days following the

onset, there was severe headache over the affected side with beginning paralysis of the sixth nerve. The pain continued for five days after the onset of the paralysis. The pain and the paralysis of the sixth nerve gradually subsided. The patient was discharged on July 8.

The blood count showed 10,400 cells, with 75 per cent polymorphonuclears.

The hemolytic streptococcus was recovered.

CASE 7.—M. L., aged 8, was admitted to the hospital on Jan. 12, 1929, with symptoms of acute mastoiditis on the right side. The mastoid was operated on on January 17. For two days following the operation, the patient had the typical distribution of pain, and on January 19, paralysis of the right sixth nerve was complete. The pain disappeared forty-eight hours after the onset of the paralysis. On February 13, the paralysis was much less marked, but was still present. The patient made an uneventful recovery and was discharged from the hospital on February 14.

The hemolytic streptococcus was recovered.

CASE 8.—S. M., a boy, aged 12, was admitted to the hospital on April 8. He presented the signs of an acute mastoiditis on the right side, and was operated on on April 18, with breaking down of the mastoid. The mastoid was pneumatic. The day following the operation, there was a painless onset of complete paralysis of the sixth nerve, which gradually subsided thirty-eight days after the operation. The syndrome was entirely painless throughout. He was discharged on May 26.

The blood count showed 18,000 cells, with 82 per cent polymorphonuclears.

The hemolytic streptococcus was recovered.

CASE 9.—C. G., aged 9, was admitted to the hospital on April 12. After a double mastoid operation had been done for purulent, diseased mastoids, the signs and symptoms of an otitic sepsis developed. This was accompanied by the typical pain in the right side of the face and eye. At this time the right sixth nerve became paralyzed. A sinus and jugular operation was done. A thrombosis was found in the sigmoid sinus and in the superior petrosal sinus. The paralysis of the sixth nerve gradually disappeared, and the patient was discharged well on May 18.

The hemolytic streptococcus was recovered.

CASE 10.—R. M., aged 10, was admitted to the hospital on April 12. Following an attack of measles, there was a discharge from both ears for four weeks. Pain had persisted in the right eye and the head for three weeks before admission, gradually diminishing until a week before admission, when the patient had a complete paralysis of the right sixth nerve. There was no evidence of involvement of the mastoid. No operation was done. The patient was discharged from the hospital on June 1, with resolving ears but persisting paralysis of the sixth nerve.

The hemolytic streptococcus was recovered.

CASE 11.—S. S., aged 8, was admitted to the hospital on June 16. For several days before the mastoid operation done on the day of admission, pain developed in the left eye and left side of the face. A few days following the mastoid operation, the sixth nerve became paralyzed. The typical pain persisted for many days after the onset of the paralysis of the sixth nerve but gradually diminished, and the patient was discharged on July 4, with a recovering sixth nerve which under observation was completely restored.

The hemolytic streptococcus was recovered.

CASE 12.—J. G., aged 11, was admitted to the hospital on Dec. 18, 1929. Three and one-half weeks before admission, there was a spontaneous perforation of the

left drum. The day before admission, there was complete paralysis of the sixth nerve on the left side. There were no signs of mastoiditis. The drums gradually healed, and the sixth nerve gradually recovered. There was no operative indication. The patient was discharged on Jan. 19, 1930, with a recovered sixth nerve.

CASE 13.—S. A., aged 4, was seen in the office of one of us (I. F.) on Feb. 5, 1930. Five weeks before myringotomy with diffuse discharge, there had been frontal headache at varying intervals. Three days before I saw the patient, paralysis of the sixth nerve was complete. One week before, there had been headache and vomiting for one day. The patient had a surgical mastoiditis, and was referred to the surgeon who sent him. The patient made an uneventful recovery.

CASE 14.—N. G., a boy, aged 9, was admitted to the hospital on March 16, 1927. On the day of admission a simple mastoid operation was done. Pus was found at the tip. The mastoid was pneumatic. For seventeen days following the operation the temperature was normal. Ten days after operation, the patient complained of pain in the left eye, which was promptly followed by paralysis of the sixth nerve on the affected side. The pain in the eye persisted throughout, and on the seventeenth postoperative day there was a fulminating meningitis in which the hemolytic streptococcus was found. The patient died on April 20. On postmortem examination, there was an osteitis of the apex of the petrous pyramid with a large epidural abscess and a diffuse purulent leptomeningitis.

The hemolytic streptococcus was recovered.

CASE 15.—E. M., aged 28, was admitted to the hospital on Aug. 30, 1924. For three weeks before admission, the patient had had excruciating left hemicrania. On the day of admission, a simple mastoid operation (left) was done. Extensive destruction of the bone was found. On the third postoperative day, the left sixth nerve was completely paralyzed, and on the fifth day the patient had a fulminating meningitis. The patient died on September 7.

Pneumococcus type IV was recovered.

THE BLOOD IN THE VARIOUS ANGINAS*

JOHN J. SHEA, M.D.
MEMPHIS, TENN.

Hematology is undergoing radical changes. The relationship of the anginas and the hematopoietic system is uncertain, for in one instance an infection of the throat may be the initial lesion and in another the terminal manifestation of a hematopoietic disease. The medical student of ten years ago was taught a theoretical classification of the diseases of the blood which was not practical when applied in the routine of medicine. Today, one seeks a better knowledge of the possibilities of the hematopoietic response to infection, and this knowledge can best be obtained through a closer study of the observations on the blood in the anginas.

In 1907, Turck [1] described a hematopoietic disease that has become known as agranulocytic angina. In 1922, Schultz [2] again drew attention to the disease, and since then many cases have been reported in the literature. I wish to report one true case, and two cases, one resulting from neoarsphenamine and the other of an unknown origin, the blood pictures of which were those of agranulocytic angina.

The older physiologists considered the blood as a special form of connective tissue having a fluid matrix. The modern conception is that the blood is a mixed secretion the formed elements of which are the result of parent cells living outside the blood stream. These hematopoietic tissues differ in the embryo and the adult. The fetal blood is formed as a result of the activities of the blood islets in the yolk sac, the primitive liver and the reticulo-endothelial system. The capillaries of the formative bone-marrow, the lymphoid follicles of the spleen and other lymphoid structures next add to this natal blood. The process changes gradually, until in adult life there exist groups of parent cells outside the blood stream, with four major hemic units in the blood. From the parent myeloblasts come the granular cells, which are the polymorphonuclear leukocytes and the monocytes; from the lymphoblasts are derived the lymphocytes, and from the normoblasts, the red blood cells.

* Submitted for publication, May 26, 1930.

* Read before the American Laryngological Association, Swampscott, Mass., May 22-24, 1930.

1. Turck, W.: Septische Erkrankungen beim Verkummerung des Granulozytensystems, Wien. klin. Wchnschr., 1907, p. 157.

2. Schultz, W.: Ueber eingenartige Halserkrankungen: (b) Gangraneszierende Prozesse und Defekt des Granulozytensystems, Deutsche med. Wchnschr. **48**:1495, 1922. Varsé: Pathologisch-anatomische Befunde bei der Agranulozytose, Deutsche med. Wchnschr. **48**:1496, 1922.

The bone-marrow houses an emergency supply of polymorphonuclear leukocytes, and can deliver them into the circulation when needed. If the surplus is consumed and the newly formed cells are not sufficient for the demand, immature forms will be found in the circulation. The most common causes for the presence of immature cells in the blood are: (*a*) hemorrhage, (*b*) diseases that destroy blood cells, (*c*) various substances that act as stimulants to the hematopoietic system and (*d*) hyerplasia of the hematopoietic tissues.

The infections of the throat are characterized by the influence they exert on the hematopoietic system, and belong to the third class. On the other hand, there are primary diseases of the blood that produce lesions in the mouth, and it is difficult to separate the borderline cases.

The laryngologist has an opportunity to study the relationship of the anginas and the hematopoietic response in the following diseases: agranulocytic angina, acute infectious mononucleosis, Vincent's angina with a lymphoid leukocytosis and acute lymphadenosis.

The anginas are capable of producing four types of changes in the blood: (1) polymorphonuclear leukocytosis, as seen in follicular tonsillitis and septic throats; (2) mononuclear leukocytosis, as occurs in infectious mononucleosis; (3) leukopenia with a decrease of granulocytes—agranulocytic angina— and (4) leukocytosis with a relative hyogranulocytosis, as seen in the patients suffering from a generalized Vincent infection.

AGRANULOCYTIC ANGINA

Today, laryngologists see more cases of agranulocytic anginas either because the disease is more frequent or because the laryngologist is more alert. There are two varieties, one that ends fatally and is most frequently seen in middle-aged women, and another that is only a complex. The etiology of the disease is not definitely known. Various observers have isolated the following organisms from lesions of the throat in these cases: *Bacillus pyocyaneous,* hemolytic and nonhemolytic steptococci, staphylococcus and Vincent's bacteria. The toxin that causes the malady is capable of paralyzing the hematopoietic system, especially the bone-marrow, where the myeloblasts are producing granular units. The action of the toxin is the same as that of massive doses of roentgen rays on the long bones. The initial sore throat is the usual portal of entrance of the infection, but later other mucous membranes, as the intestines and vagina, present lesions. The throat picture depends on the amount and the location of the lymphoid tissue that it contains, and if the patient has retained her tonsils they will be found to be covered with an exudate. Thereafter, the progress of the disease is that of a general infection which ends fatally. The true case that I shall report was clinically diphtheria.

Strangely, the glandular system does not present uniform enlargements, as only the glands immediately adjacent to the lesion enlarge. The logical treatment in these cases is to supply the deficiency, which is blood, and to stimulate the hematopoietic tissues to renewed activity. Lymphocytes are relatively increased though absolutely decreased. The red blood cells, platelets and hemoglobin remain within normal limits until the terminal stage, when they take on the picture of secondary anemia. The bleeding and coagulation time remain normal, and the blood of these patients type and match readily when a donor is being sought for transfusion. The growth reported on the blood cultures has not been consistent for any organism.

Case 1.—*Clinical History.*—Mrs. M., a white woman, aged 51, a housewife, was seen in consultation with Dr. Henry G. Rudner at the Baptist hospital on May 6, 1929,[3] at which time she complained of weakness, sore throat and fever. One sister had died with a blood dyscrasia (myelogenous leukemia). One uncle had died of carcinoma of the throat, and one of her grandmothers had died of a malignant condition of the eye. The patient had had frequent attacks of tonsillitis during childhood; the right tonsil had been tonsillotomized at the age of 21, with good after-results. During the past summer, she had had "malaria" but had not taken quinine. Eight months before examination she had neuritis and pyorrhea, and her remaining teeth were extracted.

Three weeks before examination she had fever with no localizing symptoms. Quinine was administered, but was discontinued because of urticaria. One week later a severe sore throat developed, and the family physician gave her diphtheria antitoxin, as both tonsils were covered with a pseudomembrane. For one week her throat was sore and painful, and the fever rose each evening as high as 104 F. A few days previous to admission she had diarrhea and pain in her knees.

General Examination.—The general appearance was that of an acutely sick woman with an icteroid tinge. There was herpes on the nose and lips There were no teeth, and the alveolar processes were normal. The tongue was heavily coated with a white fur. The pharynx was infected. There was a necrotic ulcer on the anterior pillar of the left tonsil, and the posterior pillar of the same tonsil was displaced by a swelling from behind which was probably a gland. The tonsils were clean, but had a cadaveric appearance. The anterior glands of the neck were palpable and tender.

There were numerous bronchial râles. The heart was normal. There was a generalized tenderness of the abdomen, but the liver and spleen were not palpable. The genitalia showed marked irritation with an erosion on the left vulva. The knees were tender but not enlarged, with normal mobility. There was a small erosion of the rectum.

Examination of the blood showed: red cells, 3,240,000; white cells, 1,200; polymorphonuclears, 10 per cent; large lymphocytes, 10 per cent, and small lymphocytes, 80 per cent.

Urinalysis gave negative results.

Smears from the lesion of the throat were rich with the bacteria of Vincent's angina. A culture made from the same region was negative for diphtheria bacilli.

3. Case reported by Dr. H. G. Rudner before the Medical Section of the Southern Medical Association, Miami, Fla., 1929.

Progress.—The lesions of the mouth became more lymphocytic, and similar lesions appeared in the rectum. Severe diarrhea ensued from similar intestinal lesions, and the patient gradually grew weaker and died.

Treatment.—The treatment consisted of three whole blood transfusions averaging 200 cc. Deep roentgen therapy was applied to the tonsils, the glands of the neck, the pelvic girdle and the long bones. A blood count was made daily, and

Fig. 1 (case 1).—Photomicrograph of tonsils, showing atrophy of the lymphoid elements.

descended as low as 500 leukocytes, with an average of 10 per cent of granular cells.

Autopsy.—Autopsy revealed that the bone-marrow was aplastic and consisted mostly of fat, with a few small areas of marrow cells, consisting chiefly of lymphocytes and other mononuclear cells. Microscopically, the tonsils showed atrophy of the lymphoid structure (fig. 1).

Arsenical Poisoning with an Agranulocytic Blood Picture.—The intravenous administration of arsenical compounds may at times depress

the hematopoietic system and present a symptom complex resembling acute agranulocytic angina.

CASE 2.—Mrs. D. P., aged 26, who was seen in consultation, had an acute condition in the throat, a fever and a rash previously diagnosed as "measles."

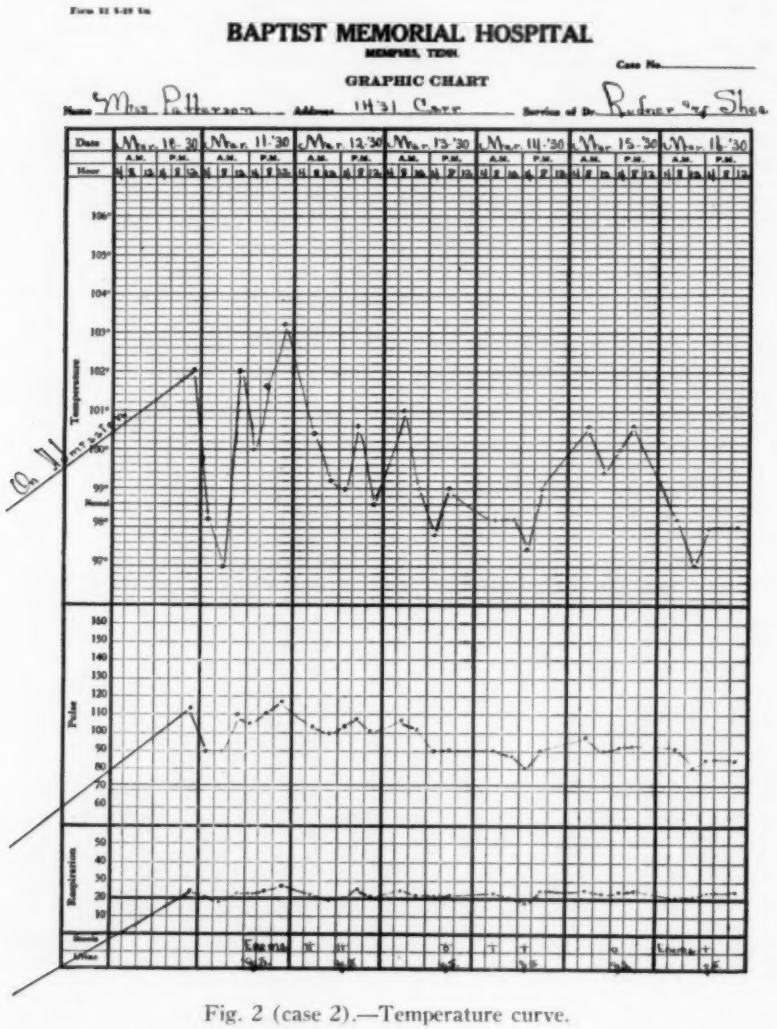


Fig. 2 (case 2).—Temperature curve.

During a physical examination, her blood was found to give a 4 plus reaction to the Wassermann test, and arrangements were made for a course of neoarsphenamine. Three days after the second dose a fever of 104 F. developed, with a macular rash.

On admission to the hospital three days later she still had a slight rash, and her blood count was reported as follows: red cells, 3,300,000; hemoglobin, 60 per cent; white cells, 4,400; polymorphonuclear neutrophils, 2.5 per cent; polymorpho-

nuclears eosinophils, 2 per cent; small lymphocytes, 81.5 per cent; large monocytes, 14 per cent. Treatment consisted of one blood transfusion of 300 cc. of whole blood, the intravenous administration of 7½ grains (0.78 Gm.) of sodium thiosulphate every twelve hours for three days and a supportive diet rich in vitamins.

A chart of her blood showed the gradual increase of the granular elements, and the blood count on discharge was: white cells, 9,100; polymorphonuclears,

TABLE 1.—*Observations on the Blood in Case 2*

Date, March, 1930	Hemoglobin, per Cent	Erythrocytes, per C.Mm.	Leukocytes, per C.Mm.	Polymorphonuclear Neutrophils, per Cent	Eosinophils, per Cent	Basophils, per Cent	Small Lymphocytes, per Cent	Large Lymphocytes, per Cent	Large Mononuclears, per Cent	Transitionals, per Cent
11	60	3,500,000	4,900	2.5	2	..	81.5	..	14	..
12	..	4,100,000	4,100	2	1	..	56	3	36	2
13	..	.....	4,300	4	..	..	64	2	30	..
14	..	4,100,000	4,650	8	..	..	68	1	23	..
15	..	.....	4,550	13	..	..	61	1	24	1
16	..	.....	4,550	22	..	1	62	..	15	..
17	..	.....	5,000	36	..	..	64	..	10	..
18	..	.....	5,200	37	..	..	58	..	5	..
19	..	4,900,000	5,100	49	..	..	42	..	9	..
20	..	.....	6,500	52	..	..	42	..	6	..
21	..	.....	7,250	60	..	..	36	..	4	..
22	..	.....	7,500	62	..	..	32	..	6	..
23	..	.....	9,100	74	..	..	25	..	1	..

TABLE 2.—*Observations on the Blood in Case 3*

Date, March, 1930	Hemoglobin, per Cent	Erythrocytes, per C.Mm.	Leukocytes, per C.Mm.	Polymorphonuclear Neutrophils, per Cent	Small Lymphocytes, per Cent	Large Lymphocytes, per Cent	Large Monocytes, per Cent	Lymphoblasts, per Cent
22	80	4,000,000	1,800	1	97	..	2	..
23	..	.....	2,150	3	96	..	1	..
24	..	.....	1,550	2	90	..	4.5	3.5
25	..	3,700,000	1,550	3	82	8	5	2
Transfusion								
27	..	.....	3,950	28	60	3	8	1
28	..	4,600,000	4,000	41	52	2	5	..
29	..	.....	4,300	48	40	5	7	..
30	..	.....	6,300	58	33	..	9	..
31	..	4,500,000	6,900	58	40	..	2	..
April 1	..	.....	11,100	72	24	..	4	..
April 2	85	4,500,000	10,500	73	20	..	7	..

75 per cent; small lymphocytes, 25 per cent; large monocytes, 1 per cent. The course of the blood is shown in table 1.

The temperature curve is shown in figure 2.

Agranulocytic Angina Complex.—The following case is one of agranulocytic angina, of unknown cause.

CASE 3.—W. S. M., a man, aged 60, was admitted to the hospital convalescing from pneumonia. He had a bilateral acute maxillary sinusitis which improved under simple irrigation, and he was ready for discharge when acute angina with a rising temperature developed. A small ulcer was visible on the left tonsil, from which many fusiform bacilli and spirochetes of Vincent were obtained. The

blood picture was a revelation: white cells, 1,800; polymorphonuclear neutrophils, 1 per cent; small lymphocytes, 97 per cent; large monocytes, 2 per cent. The granular elements were reduced to 3 per cent, showing that the hematopoietic tissue, the function of which is their production, had been paralyzed. Table 2

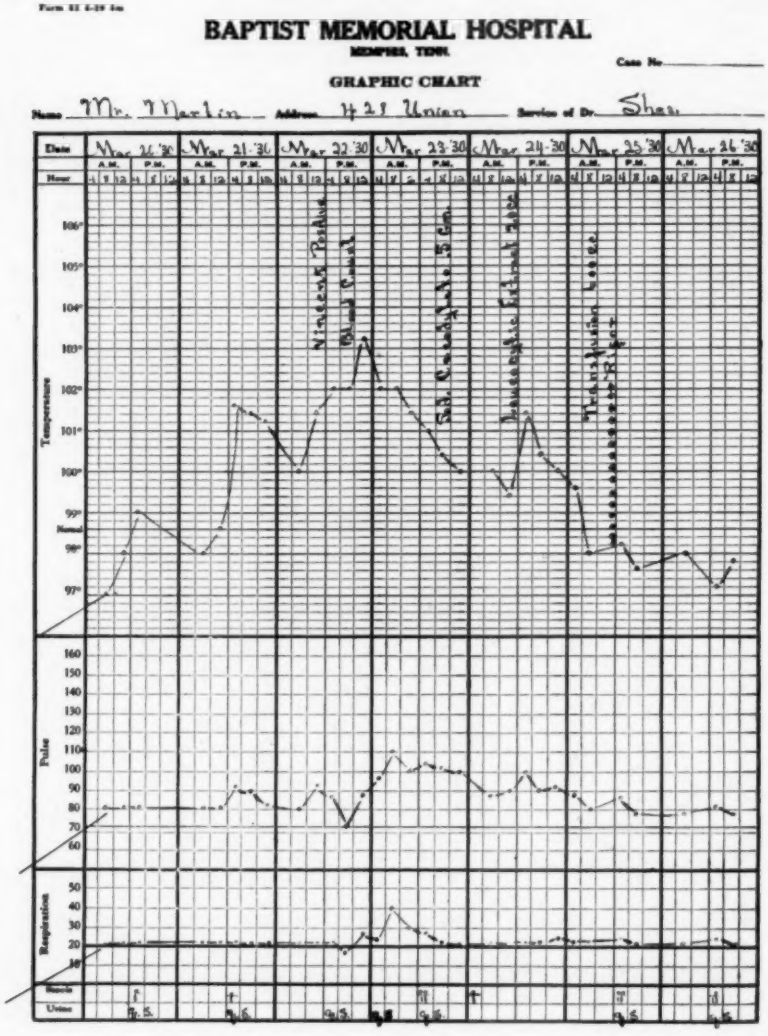


Fig. 3 (case 3).—Temperature curve during the course of acute angina.

and figure 3 demonstrate the course of the observations on the blood, and the temperature curve.

The toxin that paralyzed the hematopoietic system was not that of the Vincent organisms. The next case is typical of the fatal type of this disease.

VINCENT'S ANGINA

CASE 4.—E. L., a man, aged 24, was admitted to the hospital with a severe case of angina following a prophylactic dental treatment. His gums were ulcerated and swollen, and the glands of the neck were enlarged. Hemorrhages from the stomach and intestines, the result of a severe toxic condition, caused his death five days after admission. Smears from the gums were positive for Vincent's organisms, and every visible lymphoid tissue of the throat was ulcerated. The observations on the blood are shown in table 3.

In these cases of Vincent's angina the predominating cell is a mature small lymphocyte, and in the less rapid cases their percentage will be over 90.

INFECTIOUS MONONUCLEOSIS

The monocytes are normally on the increase during the second stage of leukocytic response to infection, and in chronic malaria the percentage of monocytes is from 6 to 10.

TABLE 3.—*Blood Count in Case 4*

Date, March, 1930	Hemoglobin, per Cent	Erythrocytes, per C.Mm.	Leukocytes, per C.Mm.	Polymorphonuclear Neutrophils, per Cent	Small Lymphocytes, per Cent	Large Lymphocytes, per Cent
11	46	2,700,000	40,200	3	44	53
14	38	2,100,000	37,800	3	49	48

In 1889, Pfeiffer described an acute febrile disease characterized by an angina with enlarged glands, and having a blood picture in which the monocytes and lymphoid leukocytes were increased. Many names have been assigned to this condition: glandular fever, infectious mononucleosis, acute benign lymphoblastosis and acute lymphadenosis.

The disease is the result of some toxin that stimulates the hematopoietic system, the resulting new cell produced being a mononuclear leukocyte.

There are three possibilities: lymphocytes, monocytes or myelocytes. The third is a possibility, but I have never seen an acute case of angina with an increase of the myelocytes, but the other two types are more common than is appreciated.

Lymphocytic Leukosis Following Angina.—The spreading of Vincent's disease throughout the United States by the return of the soldiers from France has increased the frequency of leukosis. Vincent's disease is usually a local pathologic entity, but occasionally it may excite a systemic response, and if this response is hemic, a lymphocytic leukosis will result. The number of leukocytes may reach from 30,000 to 40,000, and the percentage of lymphocytes may be over 90. These patients usually die unless carefully treated. These cases differ from those of acute lymphadenosis, of which the following is an example.

ACUTE LYMPHADENOSIS

CASE 5.—Mrs. T. S. W., aged 23, formerly an operating room nurse, was admitted to the hospital with an acute glandular enlargement. The blood count showed: white cells, 399,000; small lymphocytes, 16 per cent; large lymphocytes, 6 per cent; polymorphonuclears, 78 per cent. One month previously she had undergone a minor gynecological operation at which time she was thoroughly examined and found to be in average health. The progress of the disease was rapid, and the patient died one month after admission.

Are these small cells stem cells or mature small lymphocytes? In this case the granular cells responded in the beginning, and the first differential count of 78 per cent is in marked contrast with the 3 per cent of the last count. A thorough study of the patient was nonproductive of any knowledge of the etiology of her disease. The mouth was carefully watched for Vincent's infection, but remained clean though very painful during the last week of life. The terminal infection in the mouth and the lesions in the throat were due to hyperplasia of the lymphoid tissue in the mucous membrane. The tongue became dry and coated with old

TABLE 4.—*Blood Count in Case 5*

Date	Red Cells	Hemoglobin, per Cent	White Cells	Small Lymphocytes, per Cent	Large Lymphocytes, per Cent	Polymorphonuclear Neutrophils, per Cent	Lymphoblasts, per Cent
12/22/28.....	.....	..	399,000	16	6	78	..
12/26/28.....	.....	..	202,000	42	29	27	2
12/30/28.....	.....	..	352,000	50	30	15	5
1/14/29.....	1,600,000	25	205,000	88	5	4	3

blood, and the patient complained of a burning of the tongue similar to that suffered by a patient with pernicious anemia. The glands of the neck remained the same size and consistentcy throughout the course of the disease.

Mononucleosis.—A febrile condition starting with angina and accompanied by the usual headaches and malaise, but the blood study of which shows a relative and absolute increase of monocytes, is the true "infectious mononucleosis." It occurs most frequently in the young adult, and females are more susceptible than males. The etiology is nonspecific, and the course is benign. The oxydase stain will differentiate the monocytes.

The glands of the neck are enlarged, but not strikingly so, and the spleen is palpable. The first study of the blood with Wright's stain will merely show a relative and absolute increase of the mononuclear leukocytes, but the oxydase process will bring out the granules in some of the cells, thereby separating the lymphocytes and the large mononuclears. The symptoms subside gradually, but the blood picture remains "mononuclear" for months. The lesions in the throat in these cases vary from slight ulceration to extensive inflammations, and the bacterial study of direct smears and cultures gives no help. The presence of Vincent's organism in the direct smear is common if there is an anaerobic recess,

as under a membrane covering a slough. The total white blood cell count does not increase to an alarming degree, and remains under 20,000.

CASE 6.—Last year I saw in consultation a young married woman with a persistent angina; the blood picture showed: red cells, 3,800,000; hemoglobin, 75 per cent; white cells, 12,100; polymorphonuclear neutrophils, 32.5 per cent; polymorphonuclear eosinophils, 0.5 per cent; small lymphocytes, 58 per cent; large monocytes, 7.5 per cent; six months later the count was: red cells, 4,200,000; hemoglobin, 75 per cent; white cells, 5,800; polymorphonuclear neutrophils, 50 per cent; polymorphonuclear eosinophils, 1 per cent; small lymphocytes, 45 per cent; large monocytes, 4 per cent.

When the patient was first seen, a smear from the lesion on the tonsil was rich in the organisms of Vincent, and it was feared that the case would progress to lymphoid leukocytosis, but a study of the blood with an oxydase stain showed 7.5 per cent of large mononuclear cells, and it was then known that the case was in the benign class, acute infectious mononucleosis.

CONCLUSIONS

1. The toxins of the various anginas may stimulate or paralyze the hematopoietic system.

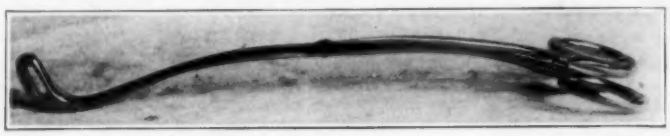
2. A closer study should be made of the blood of patients with angina.

Clinical Notes

AN ADENOID SPONGE-HOLDER*

LEO SCHWARTZ, JR., M.D., NEW YORK

The accompanying illustration shows a sponge-holder for applying pressure at the anatomic location of the adenoids. It is shaped to conform to the normal curvatures of the mouth and the nasopharynx, thereby enabling one to apply force at the proper location. The instrument should be applied in a manner similar to



that used in inserting the adenotome. It minimizes the trauma to the pillars, palate, uvula, pharynx and nasopharynx. For this reason, it has a distinct advantage over the straight and other inadequately shaped sponge-holders hitherto in use.

The instrument has been manufactured for me by Pfau's American Instrument Company of New York.

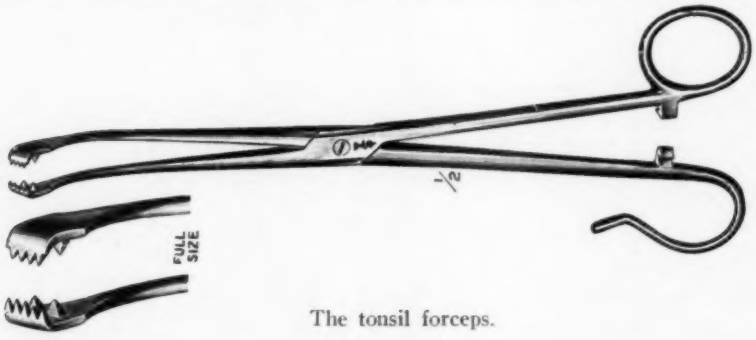
215 West 75th Street.

* Submitted for publication, April 15, 1930.

NEW TONSIL FORCEPS*

MURRAY BERGER, M.D., NEW YORK

The reason that there are so many tonsil grasping forceps on the market is that there is none suitable for all tonsils (babies; adults) which at the same time enables one to grasp small tonsillar tags. I have tried to embody certain features in the new instrument that will be able to cope with the foregoing conditions.



The tonsil forceps.

The chief advantages of this instrument are: 1. There are two sets of teeth just behind the jaws, the advantage being that one can take hold of a soft tonsil without tearing it. Moreover, small pedicles can be grasped and held, thus doing away with a second instrument. 2. The instrument is slender and is well made. 3. It has a narrow open handle, which enables one to slip a small wire loop over it. 4. There is a single tight lock.

Kny-Scheerer Corporation, of 580 5th Avenue, New York, developed this instrument.

144 West 86th Street.

* Submitted for publication, May 7, 1930.

Progress in Otolaryngology

A Summary of the Bibliographic Material Available in the Field of Otolaryngology

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ACUTE AND CHRONIC OTITIS MEDIA AND SINUS THROMBOSIS *

SAMUEL J. KOPETZKY, M.D.
NEW YORK

A careful perusal of the literature for 1929 on acute and chronic otitis media and sinus thrombosis impresses one with the fine caliber of most of the articles. As has been my custom in previous summaries, I have selected for discussion only those reports that deserve special consideration because of the excellence of their material or the originality of the ideas presented.

From the general tone of the articles studied, there is evident a tendency to reevaluate certain fundamental principles. Where definite pathologic and anatomic changes have been observed, a better understanding is sought, and has to a certain extent been achieved, of how and why those changes came to be present. This is largely due to the classic work of Wittmaack,[1] an understanding of which clarifies much in the mechanics of pathology that has heretofore been obscure.

It is interesting to note that Wittmaack's theories of otitic infection, which are based purely on anatomic observations, are beginning to be substantiated by workers in other fields. It might not be amiss to outline Wittmaack's ideas briefly. From a series of microscopic studies, he has determined that otitis media neonatorum, of both the catarrhal and the suppurative type, produces an unquestionable change from the normal in the tympanic mucosa, which remains throughout life. When the normal tympanic mucosa is composed of a low, cuboidal epithelium covering a thin layer of subepithelial connective tissue, otitis media neonatorum catarrhalis results in failure of the embryonal myxomatous subepithelial connective tissue to contract and destroys areas of the epithelium which are later replaced by high, cylindric epithelium from the eustachian tube. Otitis media neonatorum purulenta, on the other hand, causes a rapid fibrotic contraction of the subepithelial layer and a replacement of the epithelial covering by scar tissue. In addition to

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* Submitted for publication, July 15, 1930.

* From the Oto-Laryngological Department of Beth Israel Hospital.

1. Wittmaack, K.: Ueber normale und pathologische Pneumatization, Jena, Gustav Fischer, 1918.

these changes, the infantile infections under discussion have an influence on the pneumatization of the mastoid process which determines the type of the latter in later life. In other words, whether a mastoid process will be pneumatic, sclerotic or diploic is determined by the foregoing factors. For a more detailed survey of this problem, the reader is referred to the original work of Wittmaack on the subject.[1]

As a natural corollary to this newly gained knowledge, the significance of operative observations and the indications for operative intervention are being revised, and a sharper definition of surgical therapy is in development. This work lends support to the belief that the identity of the bacterial invader of the middle ear tissues has less influence on the resultant pathologic process than the anatomic structure and development of the tissue which is the host; in other words, the local reactions of the tissue depend more on its structural constitution than on the kind of bacterial flora invading it.

ACUTE PURULENT OTITIS MEDIA

General Topics.—An interesting piece of experimental work was performed by Hesse.[2] He sought to determine what influence diabetes has on the course of an otitic infection. As he pointed out, while the clinical experience of some otologists has been that diabetes tends to protract the course of acute otitis and increase its severity in other ways, there are other observers who take the opposite view and claim that the course of an otitic infection in a diabetic person is just the same as that in a normal person.

Hesse's experiments were conducted on depancreatized dogs that were kept alive on insulin from two to four weeks. After the diabetes was thoroughly established, a culture of virulent organisms was injected into the middle ear of the animal. The middle ear of a healthy dog was infected in the same manner at the same time. On the whole, the otitis media in the diabetic dogs proved to be more protracted and more severe, on microscopic tests, than the corresponding aural infection in the animal used as a control. For instance, one dog from which the pancreas was removed and in which the aural infection was permitted to run for thirteen days before he was killed, showed the following changes: There was a marked swelling and inflammation of the entire mucosa of the middle ear. The mucosal vessels were markedly dilated. The entire tympanic cavity was filled with profuse purulent secretions which were especially marked in the region of the stapes. In addition, and this is important, there were areas in the bony walls where osteoclasts were present in considerable numbers. The control dog of this

2. Hesse, W.: Experimentelle Untersuchungen zur Frage des Einflusses eines bestehenden Diabetes auf den Verlauf der akuten Otitis media, Ztschr. f. Hals-, Nasen- u. Ohrenh. **24**:512, 1929.

experiment, which also had a discharge of thirteen days' duration, showed a thickening and inflammation of the tympanic mucosa only in the lower anterior portion of the bulla tympani. The secretion was scanty, and the degree of inflammatory reaction was much less marked than in the diabetic dog. Furthermore, there were no osteoclasts in the bony walls.

These experiments of Hesse seem to have settled beyond question the fact that the course of otitis media in a diabetic is much more severe than in a nondiabetic person. It is a clinical fact that diabetes produces what is commonly termed the painless type of acute coalescent mastoiditis. Heretofore it was thought that this lack of symptoms was the essential characteristic of the disease. Hesse showed that the diabetic patient with an aural infection actually presents a greater amount of destruction and inflammatory reaction than ordinarily occurs in acute otitis media. I have observed that the time during which expectant treatment may safely be carried on is much less in diabetic than in nondiabetic persons, and I have repeatedly drawn attention to the gravity of otitic infection in these patients and warned against the continuation of expectant treatment until the classic signs of acute mastoiditis appear.[3] Hesse's observations give substantial support to this position.

Hesse [4] also reported on the bacteriologic observations in 276 cases of suppuration of the middle ear. According to him, the severity of the infection depends not so much on the virulence of the bacteria as on the degree of bodily resistance present. He also corroborated Wittmaack's views with respect to the influence of the type of tympanic mucosa on the course of aural suppurations. He further stated that while *Streptococcus mucosus* is very malignant in most cases, it showed no especially malignant tendencies in infections occurring during the first two years of life.

It is difficult to understand on what these conclusions are based. The severity of all infection is a matter of proportion between bacterial virulence and bodily resistance. Hesse's report does not indicate any exact measurement of bodily resistance and he has not ascertained the virulence of the organisms in his 276 cases. As a matter of fact, there are few real data that can be used clinically to estimate bodily resistance or the virulence of a given infection. The remarkable work of Hadjopoulos [5] in estimating the virulency of infection is still in its

3. Kopetzky, S. J.: Otologic Surgery, ed. 2, New York, Paul B. Hoeber, Inc., 1929, p. 106.

4. Hesse, W.: Beiträge zur Bakteriologie und Klinik akuter Mittelohreiterungen, Ztschr. f. Hals-, Nasen- u. Ohrenh. **22**:372, 1929.

5. Hadjopoulos, L. G.: A Study of Streptococci in Relation to Pathogenicity and Sugar-Fermenting Properties, J. Lab. & Clin. Med. **15**:539, 1930.

beginnings. The answer from the laboratory is too slow; by the time the latter is ready to give a report, the case has either terminated, clinically, or complications have developed.

Vogel [6] reported fifty cases of aural suppuration due to *Streptococcus mucosus-encapsulatus* in which he performed an operation. Twenty-five of these cases showed the typical progressive course of this type of infection without any symptoms. In twenty-seven, endocranial complications were present. The enormous percentage of intracranial lesions associated with this type of infection should warn one of the danger that exists when it is found to be the causative factor in aural suppuration. Hesse's observation that this organism shows no particularly malignant tendencies in infections occurring during the first two years of life requires clinical substantiation. I usually perform an early and thorough operation on all adults presenting this organism, since I feel that one must go beyond the lesion if the life of the patient is to be saved. Because this organism fails to produce marked local reactions in the tissue in which it lodges, it is difficult to outline hard and fast indications for therapeutic surgery. The younger the patient, the greater the possibility of spontaneous recovery without operation.

Hofmann [7] made bacteriologic studies of acute otitis media in children in whom the discharge from the ear was fetid. He observed that these cases showed a tendency toward a protracted course. While he isolated anaerobic saprophytes like *Proteus communis, pyocyaneus* and *B. coli,* he found that the fetor was due mostly to a pseudodiphtheritic organism. He is of the opinion that it is the latter which tends to cause not only the fetor, but the long duration, of the otitis. Without questioning Hofmann's observations, his conclusions cannot be accepted in their entirety. It is well established that secondary infections add elements to all otitic infections that prolong the discharge. In the earlier literature this factor was taken as a sign of chronicity. Today there is an advance beyond this. The work of Wittmaack, to which reference has already been made, shows other elements that play a far more important rôle than secondary bacterial infection in the production of chronicity.

Berberich [8] conducted experiments in man to determine the significance of bulging in Shrapnell's membrane. He studied cases that presented this sign and noted that after paracentesis of the pars tensa the

6. Vogel, K.: Erfahrungen über Mucosus-Otitis, Ztschr. f. Hals-, Nasen- u. Ohrenh. **22**:357, 1929.

7. Hofmann, L.: Die Keimflora fötider akuter und chronischer Otitiden, Ztschr. f. Hals-, Nasen- u. Ohrenh. **24**:423, 1929.

8. Berberich, J.: Die Bedeutung der epitympanalen Vorwöblung des Trommelfells, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **122**:15, 1929.

bulge in Shrapnell's membrane collapsed. He agreed with Kümmel that the bulging of Shrapnell's membrane is caused by a swelling of the mucosal folds in this area. In order to substantiate his clinical observations, he injected fluid into the middle ear of a cadaver and was able to produce a bulging of Shrapnell's membrane only, not of the pars tensa. This led him to believe that the pars tensa is incapable of much bulging, because of the fact that it is fixed in the annulus and in the region of the long handle of the malleus, and that the actual transfer of pressure in the middle ear is against Shrapnell's membrane, which is the only yielding portion of the drum. He accordingly stated that little prognostic value can be attached to finding a bulge in Shrapnell's membrane in cases of acute otitis media.

Shambaugh [9] reported two cases in which there were pain and a conductive deafness in the ear, and the drum was black on otoscopic examination. Paracentesis yielded a chocolate-colored fluid which oozed from the tympanic cavity, and which showed *Staphylococcus albus* on culture. Tubal inflation before paracentesis produced râles, and this led him to believe that the condition was not cystic. I have seen such a case in which there was recurrence so often that it was finally necessary to perform a total excision of the drum and ossiculectomy to give the patient relief. After that, he was relieved of his symptoms but continued to have an aural discharge.

Vignerie [10] reported a case of burn of the middle ear that is of interest. The patient, a metal worker, had hot metal poured into his left ear. Besides the trauma to the outer canal, there ensued total destruction of the drum, paralysis of the chorda tympani nerve and total nerve deafness, but the vestibular function remained normal. In all probability, the total deafness was the result of a serous labyrinthitis with its accompanying hydrops labyrinthi, the point of entry being through the round window. This would account for the retention of vestibular function and corroborates a work of Wittmaack,[11] who was able to produce such conditions experimentally by the instillation of chloroform and other chemicals into the middle ear of animals. Vignerie's report is of value from another angle. In traumatic cases (Almour [12]) it is no uncommon occurrence to find part of the labyrinth destroyed and nonfunctioning, while the remainder of the labyrinth is

9. Shambaugh, G.: The Blue Drum Membrane, Arch. Otolaryng. **10**:238 (Sept.) 1929.

10. Vignerie, M.: Un cas de brûlure de l'oreille moyenne, Ann. d. mal. de l'oreille, du larynx **48**:1125, 1929.

11. Wittmaack, K., in Henke and Lubarsch: Handbuch der pathologische Anatomie, Berlin, Julius Springer, 1928, vol. 12.

12. Almour, R.: An Interpretation of the Otological Findings in Cases of Industrial Trauma, Bull. 6, Department of Labor, State of New York, 1924.

untouched by the trauma and functions normally. In the courts where industrial cases are adjudged for compensation, it is often hard to convince the authorities on this point, because textbooks and the literature in general do not differentiate sufficiently between the labyrinthine lesions produced by trauma and those produced by disease.

Watanabe [13] reported a case of bilateral acute otitis media associated with bilateral abducens paralysis which cleared up without operation on the mastoid. The patient was 25 years old and first developed a left otitis media associated with a trigeminal neuralgia which persisted for fifty days. Twenty-five days after the onset of symptoms, a left abducens paralysis with double vision appeared which remained for twenty-five days. After this disappeared, there developed a right otitis with a similar Gradenigo syndrome, which soon cleared up, as did that on the left side. The Wassermann reaction was negative. In view of what is now known about the pneumatization of the temporal bone, a new conception is gaining ground as to the mechanics of invasion producing this symptom. Today there are otologists who look on the Gradenigo syndrome not as a complication of mastoiditis, but rather as a symptom of suppuration in the petrous pyramid. The petrosal tip of the temporal bone is often pneumatic and contains cells which communicate with and are analogous to those in the mastoid process of the bone. An infection starting in the tympanic cavity is as likely to spread to such a collection of cells in the petrosal tip, leading from the pneumatic cells at the otitic orifice of the eustachian tube, as it is to involve the cells of the mastoid process. Where such an extension takes place and there is no great involvement of the cells of the mastoid process, as a result of the lesion in the petrosal tip a reaction may be set up in the dura overlying it which involves adjacent nerves. There then occurs the so-called Gradenigo syndrome.

While on this subject, it may not be amiss to observe that the more recent knowledge of the anatomy and pathology of the petrosal pyramid of the temporal bone is leading to a reevaluation of certain clinical manifestations and gives added significance to Profant's reports, in a 1924 issue of the *Laryngoscope* and before the Section on Laryngology, Otology and Rhinology of the American Medical Association at its 1930 meeting, and to Eagleton's presentation before the Eastern Section of the American Laryngological, Rhinological and Otological Society. Otologists are coming to understand the Gradenigo syndrome as a secondary meningeal manifestation, to realize that the petrosa is a route commonly traveled by infection on its way to the meninges, that suppuration of the petrosal cells is the cause of persistent otorrhea after

13. Watanabe, T.: Ein Fall von doppelseitiger otogener Abducenslähmung, Otologia **3**:10, 1930.

simple mastoidectomy more often than was heretofore believed, and that this lesion has not received the consideration it merits. Profant has proposed the name "petrositis" for it; he reported four cases at the meeting of the American Medical Association in June, 1930. My experience is limited to nine cases, with five recoveries. Operation was performed in four cases according to a technic devised by Almour, which will be published soon.

Otitis Media Acuta in Infants.—Van Gilse [14] presented a series of microscopic preparations of the middle ear of a child, 1 year of age, who showed mild inflammatory changes in the middle ear, and in whom there was present at the same time an exudate with leukocytes in the perilymphatic spaces of the cochlea. He considered this a latent infection of the middle ear since the child presented no evidences of disease of the middle ear clinically. I do not feel that such a case should be termed latent. If one considers the easy exit afforded to pus in the middle ear of an infant by the patent eustachian tube, one appreciates the difficulty of recognizing such a disease without careful inspection of the pharyngeal orifice of the tube. The term "latent," in the foregoing sense, seems to me to be ambiguous. A lesion is present or absent, active or inactive. When "latent" is used to describe a lesion that is present but not recognized clinically, it is sometimes an admission of an inability to recognize the condition in question.

Burghi and Carlos [15] studied disturbances of digestion and nutrition in infants presenting an acute otitis media. Of 310 cases, 116 showed an otitis media, mostly bilateral. Twenty-eight cases came to autopsy, and 80 per cent of these showed otitis. One was associated with meningitis and another with sepsis. Most of these cases showed disturbances of digestion clinically, loss of weight, vomiting, diarrhea and pain on pressure over the tragus. The authors considered these observations diagnostic of a malnutrition secondary to otitic disease, and came to the conclusion that a bilateral paracentesis should always be performed in such cases to ensure recovery, for they feel that the patient will die if nothing is done. The interrelationship of otitis media and gastro-intestinal disturbances in infants has long been known. The difficulty of estimating the condition of the middle ear spaces correctly by otoscopic examination is one of the factors that prevent *intra vitam* diagnosis of suppuration of the middle ear. The work of Marriot and Alden seems to support the contentions and conclusions of Burghi and Carlos.

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14. Van Gilse, P. H. G.: Latente Mittelohrentzündung bei Menschen und Tier, Nederl. tijdschr. v. geneesk. **2**:5634, 1929.

15. Burghi, S. E., and Carlos, G.: Störungen der Verdauerung und Ernährung durch latente akute Otitis beim Säugling, An. de Fac. de med., Montevideo **14**: 1045, 1929.

O'Donnell and Meyers [16] and Timarchi [17] attempted to diagnose otitis media in infancy by means of cultures taken from the middle ear through an intact drum. The technic is to sterilize the external auditory canal carefully and then to enter the middle ear with a fine needle and draw off whatever fluid may be present. By this means they believe that uncontaminated cultures can be obtained. Timarchi obtained anaerobic organisms in all of the fifteen cases he studied. While these results are interesting in themselves, the numerical evidence is too slight to class them as anything more than an observation. The report would have been of more value if the nature of the cases presenting anaerobic organisms had been more clearly defined. For practical purposes, a piece of research detached from its clinical background is sterile.

Acute Purulent Otitis Media in Infectious Diseases.—Goldenberg [18] recorded that 199 of 702 cases of scarlet fever presented an acute otitis media, usually unilateral. Only 6 required operation on the mastoid. He stated that since the use of serum in the treatment for scarlet fever the course of the complicating otitis has been rendered much milder. Reports are still to come from the larger institutions for contagious disease throughout the country in regard to this. Most otologists are still impressed with the fact that scarlet fever tends to produce a severe necrotic lesion in the middle ear, when aural complications occur, and they do not yet rely on serums to combat it. Nevertheless, when one remembers what occurred in diphtheria, one may expect good results from a suitable serum or antitoxin. A case in point is the report of Nussbaum,[19] who had a patient with acute otitis accompanying diphtheria who was not in any way relieved by the ordinary routine treatment of the ear. The patient presented a specific membrane on the drum from which *Bacillus diphtheriae* was isolated. The administration of 10,000 units of antitoxin effected a complete cure.

Hofmann [20] reported on 1,000 cases of pneumonia studied in the Vienna Hospital for Children. Three hundred of these presented an acute purulent otitis media. Of the very young infants with pneumonia (440), 149 showed an acute purulent otitis. In 50 young infants with

16. O'Donnell, W. S., and Meyers, C.: Otitis Media in Infancy: Its Diagnosis by Means of Cultures Taken from the Middle Ear, Am. J. Dis. Child. **38**:49 (July) 1929.

17. Timarchi, A.: Richerche battereologische sull' otite latente dell' infanzia, Boll. d. mal. d. orecchio, d. gola e d. naso. **47**:65, 1929.

18. Goldenberg, M.: Zur Klinik der Sharlachotitis, Monatschr. f. Ohrenh. **63**:1164, 1929.

19. Nussbaum, D.: Diphtheria of the Ear, Arch. Otolaryng. **10**:296 (Sept.) 1929.

20. Hofmann, L.: Zur Klinik der Pneumonieotitis im Kindesalter, Monatschr. f. Ohrenh. **63**:237, 1929.

bronchopneumonia following measles, there were 29 cases of otitis. In 94 cases of pneumonia following whooping cough, there were 29 cases of otitis. After the fourth year of life, the frequency of otitis as a complication of pneumonia became less and less. Lobar pneumonia was not associated with acute purulent otitis as often as bronchopneumonia, and when otitis did occur in lobar pneumonia it ran a much less virulent course. These observations are in agreement with those of most otologists. In a general hospital it is noteworthy that cases of lobar pneumonia are rarely complicated by an otitis, whereas acute purulent otitis is of frequent occurrence in the pneumonia which follows the infectious diseases and which is most often bronchopneumonia.

Carvados [21] made some interesting observations on the severity of acute purulent otitis media following grip. He stated that the lesion is due to a mixed infection, and that the secondary infection follows soon after the first, attacking the ear before the body has had time to develop antibodies against the grippal infection. There then occurs an infiltration of the superficial and the deep layers of the mucosa. This leads to severe pain even before an exudate is formed. Since, in such a type of inflammatory reaction the exudate first develops between the epithelium and the fibrous layer of the drum, it gives rise to the blebs on the drum that are commonly seen with this form of lesion. These blebs contain serosanguineous fluid. The symptoms persist for a long time after the drum ruptures, and there is rarely a profuse discharge from the ear. According to Carvados, the pathologic process in the mucosa of the mastoid process is identical to that occurring in the middle ear.

From a clinical standpoint, Carvados' observations are significant. During the past winter, although there was an absence of grip, those cases that presented a primary lesion in the ear or an aural complication at the outset of another illness, usually underwent a protracted and stormy clinical postoperative period, although the local signs, therapeutic indications and reaction to operation were similar to those in cases in which the otitic lesion developed later in the course of a sickness. The reason for this is the same that Carvados advanced—the failure of the body to develop antibodies before the local ear condition supervened.

Joukovsky [22] reported the case of a patient, 35 years of age, who presented a right purulent otitis media from which the Vincent organism was isolated. The case developed insidiously and presented a persistent reddish-brown discharge, which was very foul. The middle ear showed excessive granulations and polyp formation, and there was a tendency to necrosis of the bone. The condition yielded readily to a

21. Carvados, E.: L'otitie moyenne aiguë et ses complications en rapport avec la grippe, Ann. d. mal. de l'oreille, du larynx **48**:1011, 1929.

22. Joukovsky, A.: A propos de la fuso-spirillose Vincent de l'oreille, Ann. d. mal. de l'oreille, du larynx **48**:1023, 1929.

course of neoarsphenamine and bismuth. Joukovsky stated that this should be the routine treatment, and that operation is rarely necessary.

Tawse [23] reported a rare case. An acute left purulent otitis media developed in a woman, 69 years of age, and was followed by an acute mastoiditis which ran a stormy course. The tubercle bacillus was definitely isolated as the cause, but radical operation did not prevent death from meningitis. It is indeed unusual for the tubercle bacillus to cause an acute local lesion in the ear. It is customary to consider tuberculosis of the ear as belonging in the category of chronic aural suppurations. To find that this bacillus is capable of producing an acute lesion adds another factor to the knowledge of tuberculosis in connection with otology.

Incidentally, Mori [24] recommended a histologic study of the polypoid mucosa of the middle ear in all cases of suppuration which run a protracted course in order to determine definitely whether or not tuberculosis is the cause. In his four cases, histologic examination gave the typical signs of miliary tubercles, and Mori expressed the belief that this is the only positive proof of aural tuberculosis. This suggestion for the diagnosis of a tuberculous otitis should be remembered, since it offers a histologic proof which can be had earlier than the results of the usual injection into animals. In the absence of polypoid masses for the proposed biopsy, other adjacent tissue might be studied to advantage.

Drury [25] and Zanni [26] reported cases of actinomycosis of the ear. In the former's patient, the lesion developed first in the region of the ear. It progressed until the middle ear and mastoid process were attacked, and the patient died. Autopsy revealed multiple abscesses in the meninges and extensive actinomycosis of the glands.

In Zanni's patient, a man 40 years of age, an acute purulent otitis media developed after severe aural pain. Ten days after the onset, a paracentesis was performed. There developed behind the ear a painless swelling, which opened spontaneously. For a short while there was a discharge which disappeared. The discharge from the ear was white, stringy and pulsating. The meatus was swollen, and there was a granulation external to the drum. The retro-auricular swelling reappeared, again opening spontaneously, and a yellow fluid which contained small particles was discharged. There was no fever, pain or adenitis. At this time a local application of iodine caused the swelling to disap-

23. Tawse, H. B.: Acute Senile Tubercular Disease of the Left Middle Ear and Mastoid, J. Laryng. **44**:255, 1929.

24. Mori, S.: Zur Diagnose der Mittelohrtuberkulose, Otologia **2**:950, 1929.

25. Drury, D. W.: Mastoiditis-Actinomycosis, J. A. M. A. **93**:974 (Sept. 28) 1929.

26. Zanni, G.: Un caso di actinomicosi dell' orecchio medio, Valsalva **5**:287, 1929.

pear, and the lesion was completely healed in twenty days. A culture of the ear was then taken; it yielded only staphylococcus. A short time later, however, the swelling again appeared, and actinomycosis was demonstrable. The patient was put on a course of treatment with potassium iodide, administered aurally, and the ear was completely and finally dry in ten days, with no further recurrences. Zanni recommended the administration of potassium iodide as the therapy of choice. He stated that irradiation should be employed only when the former does not give results or is not well tolerated by the patient.

CHRONIC PURULENT OTITIS MEDIA

Bigler [27] cited a case of chronic tympanic mucosal suppuration with a central perforation which went on to a fatal complication without an acute exacerbation or an acute clinical picture. He found that pneumococcus was the causative organism, and also, that there was an absence of pneumatization in the mastoid process. He cautioned otologists not to accept too definitely the dogma of the safety of cases presenting a central perforation.

In support of Bigler, Miyiki [28] reported a case of right chronic purulent otitis media of fourteen years' duration in a man, 24 years of age. This patient presented a small central perforation in the pars tensa with a pulsating discharge. At operation, a cholesteatoma was found to involve the entire antrum and epitympanic space. Miyiki quoted Ulrich, who described three similar cases. Reports of this nature are apt to shake one's confidence somewhat in the accepted clinical division of chronic purulent otitis media into the dangerous and the nondangerous type purely on the basis of the type and location of the perforation exhibited. In view of recent work by Wittmaack, Steuerer, Lange and others on the influence which the type of tympanic mucosa exerts on the course of otitic infections, it is highly possible that one may have to change one's conception of the characteristics of chronic lesions that are dangerous to life. The development of a perforation is dependent on several factors, among which are the extent of the ingrowth of squamous epithelium from the canal, whether or not the destruction of the fibrotic layer by the contraction of the squamous epithelium causes the squamous and the mucosal epithelium to meet at the margins of the perforation and so to form a permanent central perforation, and whether the perforation was originally due to a necrosis of the drum or to a rupture of the latter caused by the pressure behind it. It is possible that it may be necessary to take into greater consideration, not only the

27. Bigler, M.: Beitrag zur Lebensgefährlichkeit der einfachen chronischen Mittelohreiterung, Ztschr. f. Laryng., Rhin. **18**:213, 1929.

28. Miyiki, H.: Ein seltener Fall von Cholesteatom-Otitis, Otologia **3**:102, 1930.

otoscopic appearance of the lesion, but other fundamental anatomic and etiologic factors which preceded it and predisposed the ear to a chronic condition. This remains to be done. At the present stage of knowledge, such observations as Bigler's and Miyiki's lend support to the new conception of the pathology of chronic infections of the middle ear which is being prepared in the laboratory and the dissecting room.

Several interesting factors were brought out in this year's literature in connection with cholesteatoma of the middle ear. The articles of greatest interest were those of Polvogt [29] and Ruttin.[30] The former presented the microscopic observations in the case of a young child afflicted with scarlet fever and bilateral otitis. While the left ear showed the ordinary inflammatory lesion, the right showed a typical necrotic one. The microscopic sections revealed clearly that the squamous epithelium of the external canal had already grown into the ear. Polvogt stated that this observation answers the question of just when a cholesteatomatous formation develops. He advised early radical mastoidectomy in such cases.

I am inclined to disagree with Polvogt's conclusions as far as they disagree with the observations of Wittmaack and others, who have already demonstrated that the squamous epithelium is the most important factor in the cure of chronic suppurative lesions. Cases that present an active pneumatization of the mastoid process, when attacked by the chronic purulent otitis of scarlet fever, result in an acute mastoiditis which must be eradicated surgically. The necrotic lesion continues in the middle ear, owing, in the main, to the mass necrosis of the mucosal lining of this cavity with consequent involvement of the underlying bone. An ingrowth of squamous epithelium can be seen in all these cases, and represents nature's effort to repair the damage. Whether or not a cholesteatoma will develop depends largely on the outgrowth of squamous epithelium into the antrum and the space left bare in the mastoid process by the operation. Where pneumatization is faulty and the antral space has been obliterated by a sclerosis, it is more than possible that the ingrowth of squamous epithelium will be confined solely to the tympanic cavity, and that complete healing of the suppurative process will eventually take place, because the small, contracted antrum will cover with such epithelium. Cases presenting marginal perforations that have been dry for many years are not unfamiliar to otologists, and it is this observation that leads me to disagree with the conclusions of Polvogt.

Ruttin [30] presented several cases wherein a dry cholesteatoma preceded the development of an acute purulent otitis media. He cited four

29. Polvogt, L. M.: The Pathogenesis of Cholesteatoma, Arch. Otolaryng. **9**:597 (June) 1929.

30. Ruttin, E.: Cholesteatom und Akute Otitis, Arch. f. Ohrenh-, Nasen- u. Kehlkopfh. **120**:125, 1929.

such instances in which a cholesteatoma was present in the epitympanic space and antrum and was completely separated from the tubotympanic space. He stated that these cases are developmental and depend on the type of otitis media neonatorum which was present. In the event of an infantile otitis of the catarrhal type, adhesions can completely shut off the epitympanic antral space from the tubotympanic space. In such cases, the cholesteatoma develops from a blind sac which intrudes on Shrapnell's membrane; its further growth proceeds from this point. There is rarely a pneumatized mastoid process present, since the infantile otitis in itself results in a failure of pneumatization. Later, when an acute purulent otitis media occurs in the otherwise normal tympanic cavity, the acute suppuration breaks through the barrier separating the cholesteatomatous cavity from the middle ear and a cholesteatomatous suppuration results, presenting a perforation in Shrapnell's membrane. Ruttin distinguished carefully between this type of infection and acute exacerbation of a cholesteatoma. The former responds readily to modified radical operation. His observations constitute clinical substantiation of Wittmaack's theory of the pathology of otitic disease.

Turtur [31] presented an unusual case. Following scarlet fever in the second year of life, right chronic purulent otitis media developed with polyp formation in the middle ear. Some time later a postauricular swelling appeared, accompanied by prolapse of the canal wall and tenderness over the mastoid process. After a thorough radical operation, large fungoid granulations arose from the depths of the wound, reappearing repeatedly after removal. At first microscopic examination yielded no evidence of malignancy. One month after operation, however, typical lymphosarcoma was seen in sections. The tumor spread and eventually led to death. At autopsy, it was found to be 17 cm. long and 15 cm. deep and weighed 400 Gm.

Rendu [32] reported three cases wherein sensory disturbances of taste were noted during the course of chronic otitis media. Clinically, the loss of sense was demonstrable on the affected side. He stated that this condition is more frequent than one is wont to consider, but that it is overlooked because it is not tested for.

Vlasto [33] also studied the results of paralysis of the chordi tympanic nerve in the course of chronic suppurations. He examined the sub-

31. Turtur, G.: Sarcoma dell' orecchio medio e della mastoide in una bambina di tre anni affetta da otite media purulenta cronica, Boll. d. mal. d. orecchio, d. gola e d. naso **48**:1, 1930.

32. Rendu, R.: Paralysie de la corde du tympani dans l'otitis moyenne suppurée, Rev. de laryng. **50**:310, 1929.

33. Vlasto, M.: The Chorda Tympani Nerve in Otology, Proc. Roy. Soc. Med. (Sect. Otol.) **22**:146 (Oct.) 1929.

maxillary gland on the affected side in two cases, one of an attic suppuration and the other following radical mastoidectomy. In both instances, a marked degeneration of the glandular substance was noted.

Therapy in Otitis Media Purulenta.—Kalinin and Meerowitsch [34] studied the bactericidal properties of *Bacillus bulgaricus* and arrived at the conclusion that this organism has a distinct bactericidal action on many pathogenic and nonpathogenic organisms. They recommended the use of a culture of this organism in the treatment for purulent otitis media. Tampons should be soaked in the culture and then inserted in the external auditory canal down to the drum. Their tables are interesting, showing results in various forms of acute otitis media which vary from 9 to 100 per cent cures.

Mandelbaum,[35] in attempting to obtain detailed roentgenographic plates in cases of chronic purulent otitis media by the use of iodized oil, found that a large number of the cases wherein the iodized oil had been instilled cleared up and became dry. He presented this simply as an observation, and believes that the procedure is worthy of trial. The action is probably due to the bactericidal properties of iodine, and the idea of such therapy is probably along the lines of the Lederman treatment with dry iodine powder reported at the May, 1930, meeting of the American Laryngological, Rhinological and Otological Society.

Hett, Wells and Levick [36] discussed the use of zinc ionization in the treatment for suppuration of the middle ear. In reporting their results with this method in 1,652 cases of all kinds of suppuration, they stated that 75 per cent cures were obtained. They modify their report by calling attention to the fact that cases presenting necrosis of the bone in the region of the attic are not influenced by this form of therapy. They also attributed a large percentage of the failures to a lack of proper technic.

Goldman [37] discussed the results of roentgen treatment for acute infections of the middle ear. He found that this form of therapy produces either a shortened or an abortive course. An energetic discharge of the tympanic secretion results, which seems to be due to the

34. Kalinin, A. M., and Meerowitsch, O. S.: Zur Frage der Bakteriotherapie (B. bulgaricus) eiteriger Mittelohrentzündungen, Monatschr. f. Ohrenh. **63**:427, 1929.

35. Mandelbaum, M. J.: Diagnostic and Therapeutic Value of Iodized Oil in Chronic Purulent Otitis Media and Chronic Mastoiditis, Laryngoscope **36**:156, 1929.

36. Hett, G. S.; Wells, A. G., and Levick, G. M.: Ionization in Suppuration of the Middle Ear, Am. J. Phys. Therapy **6**:375, 1929.

37. Goldman, R.: Zur Roentgenbestrahlung der akuten Entzündung der Mittelohrräume, Strahlentherapie **33**:152, 1929.

destructive effect of the x-rays on the highly sensitive lymphocytes. This reaction sets in within six hours and lasts from three to eight days.

SINUS THROMBOSIS

Law,[38] before the Austrian Otological Society, presented an anatomic specimen of a right temporal bone which showed a strongly developed emissary vein running upward and posteriorly in a sulcus 6.5 mm. in width. The sulcus of the sigmoid sinus was entirely absent; the blood from the right transverse sinus emptied into the right emissary vein and not into the jugular bulb. From the emissary, the communication was with the superficial cervical veins direct. The left sigmoid sinus was markedly developed. In addition, the right middle and posterior cranial fossae were smaller than the left. He quoted Frey and Hoffman as having reported a similar observation in 1924.

Stewart [39] reported a case of mastoiditis in a patient 51 years of age on whom a radical mastoid operation was performed. At the time of operation, the author felt that he fractured the roof of the eustachian tube. The patient died from a cavernous sinus thrombosis. Autopsy showed that in addition to the thrombosis there was a fracture in the roof of the eustachian canal. Running alongside this fracture there was a small blood sinus that opened into the cavernous sinus in front and into the superior petrosal sinus behind. Here a septic thrombus was found which yielded *Staphylococcus aureus* on culture. This sinus has been described in the literature and is known as the accessory sinus of Kelck. In a recent report on examinations of thirty-one skulls this sinus was present twenty-three times. The writer called attention to the importance of the presence of fetal blood sinuses like the one described, the petrosquamous and the petro-occipital, as pathways for the spread of infection into the endocranium.

Haymann [40] discussed otogenous systemic infection occurring without sinus thrombosis. He cautioned that no such diagnosis can be made unless autopsy has shown all the cranial sinuses to be free from thrombi. He cited the case of a girl, aged 18, who developed an acute otitis media with high temperature, severe pain and spontaneous rupture of the drum. Otoscopic examination revealed a bulging red drum with a small pulsating perforation, a profuse discharge and an exquisitely tender mastoid process. On the fifth day of the illness there was a sudden

38. Law: Demonstration des anatomischen Präparates einer Aplasie des Sinus sigmoideus, Verhandl. Osterr. otol. Gesellsch. Wien., April 29, 1929.

39. Stewart, J. P.: The Persistence of Fetal Blood Sinuses and Their Relation to the Middle Ear Spaces, Arch. Otolaryng. **10**:266 (Sept.) 1929.

40. Haymann, L.: Otogene Allgemeininfektion ohne Sinus Thrombose, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **122**:116, 1929.

chill with high fever, nausea and slight vertigo. The labyrinth was normal. Mastoidectomy revealed hemorrhagic, purulent secretions within the cells. There was fluid pus in the tip cell and in the antrum. The sinus was normal and yielded blood on puncture. A blood culture taken from the arm and from the sinus yielded *Streptococcus hemolyticus*. The spinal fluid was normal. The sinus was obliterated, the wall excised and the jugular vein tied off. The entire channel was free from thrombi and contained blood. The patient died from sepsis. At autopsy, no thrombus was demonstrable in any portion of the sinus from the point of the tied jugular to the distal end of the sigmoid sinus. The jugular bulb was normal. All other cranial vessels were normal, as well as the excised portion of the sinus. Histologic examination of the temporal bone showed that the entire mucosa of the middle ear was the seat of a marked hemorrhagic inflammation. The mucosa had lost its epithelium in many places, and the subepithelial tissue was edematous and infiltrated with leukocytes and many red blood cells. The blood vessels in the mucosa were thrombosed. In this case, Haymann was of the opinion that his own experimental work on dogs and monkeys and the animal experiments of Caldera and Fienzi had been confirmed. This work has established the fact that bacteria can penetrate the sinus wall and lead to a sepsis without producing a thrombosis in the sinus itself. He expressed the belief that the septic focus in such cases is within the middle ear and mastoid process, and not in the sinus. This bears out the observations of Körner, Beck and myself on acute hemorrhagic mastoiditis. This disease is a systemic infection which produces a sepsis from the outset without causing a sinus thrombosis.

German [41] reported two cases of sinus thrombosis which ran an afebrile course. He considered that the factors that contribute to the lack of septic clinical processes can be divided into two classes, local and general. Thus no sepsis will be present if the thrombus is solid and sterile or if, while centrally infected, it is solid at both ends and undergoing organization. The general factors that contribute toward an afebrile course consist, in the main, of a decreased resistance on the part of the patient; this is noted particularly in diabetes.

Brunner [42] differentiated two types of sinus thrombosis, that which is present at the time of the primary operation but which remains latent for a time and then makes itself evident, and that which sets in after operation. The former variety, which he terms the latent type, usually

41. German, T.: Aetiologische Betrachtungen über partielle oder gänzliche Fieberlosigkeit bei Sinusthrombose, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **122**: 73, 1929.

42. Brunner, H.: Postoperative Sinus Thrombosis, Arch. Otolaryng. **10**:217 (Sept.) 1929.

follows an acute otitis and appears clinically soon after operation. When the sinus is operated on, a localized thrombus is isolated and recovery usually ensues. The second type, which Brunner terms postoperative sinus thrombosis, usually occurs in chronic cases and makes its clinical appearance about two weeks after operation. Here a mural clot is usually obtained, and these cases do not present the hopeful outlook that the latent cases do. Brunner based his observations on 154 cases of thrombosis. Twenty-two were of the postoperative type, and 11 of these terminated fatally.

Brunetti [43] discussed otogenic thrombophlebitis of the superior petrosal sinus. In view of what Lillie reported last year, his observations are of extreme interest. He stated that a chronic suppuration in the epitympanic portion of the middle ear can erode the bone and, by direct continuity, cause a thrombosis of the superior petrosal sinus, which runs in this vicinity. Isolated at first, the lesion soon spreads to the sigmoid or the cavernous sinus; it can be associated with a Gradenigo syndrome. Owing to the venous connection between the petrosal and the apex of the petrous pyramid, the prognosis in these cases is bad, as a result of the inaccessibility of the area.

McKenzie [44] described two types of bulb thrombosis, the primary bulb thrombosis which is a direct extension from the middle ear and a secondary bulb thrombosis which is an extension of a thrombosis within the sigmoid sinus. He stated that it is possible to differentiate only by opening the sigmoid sinus. When no blood comes from below after incision of a healthy-looking sinus, the diagnosis of primary bulb thrombosis is certain. In these cases he recommended that one first try drainage by means of a catheter forced through the jugular and lavage of the bulb. He also curets the bulb from both the sinal and the jugular end.

It has been shown by McKernon that one of the essential factors in the diagnosis of a primary bulb thrombosis is a normal mastoid process. In all the cases in which I have operated, it was possible to establish the diagnosis prior to operation by the presence of sepsis, the decreasing red blood cell count and hemoglobin and the normal condition of the mastoid process, in association with a purulent middle ear.

Gondelman-Binstok [45] stated that primary jugular bulb thrombosis can be cured only by an early radical Grunert bulb operation, and that in his opinion, the patient has a chance of recovery only when the vein,

43. Brunetti, F.: La thrombophlébite otogene du sinus pétreus et du sinus longitudinal, Otol. internat. **13**:313, 1929.

44. McKenzie, D.: Thrombophlebitis of the Jugular Bulb, Proc. Roy. Soc. Med. (Sect. Otol.) **22**:73 (July) 1929.

45. Gondelman-Binstok, I. S.: Zur Frage über die Behandlung der Thromboplebitis Bulbi venae jugularis, Odessky M. J. **4**:320, 1929.

bulb and sigmoid sinus are laid open as a groove. He further stated that ligation of the jugular vein alone is insufficient. I cannot agree with this author, because the majority of patients with primary jugular bulb thrombosis on whom I have operated recovered after incision of the sinus, ligation of the internal jugular vein and suction and curettage of the jugular bulb. These cases present the danger that a continued focus of sepsis will be set up by extension into the inferior petrosal sinus or the condylar veins. I believe that the establishment of drainage of the jugular bulb is in itself insufficient for the cure of this lesion. If one is fortunate in recognizing the condition early and performing an operation before the thrombosis has extended into the communicating channels, the recovery is spectacular. On the other hand, once the septic focus has extended into the inferior petrosal or the condylar veins, it is impossible to eradicate the lesion thoroughly by operation even when a bulb operation is performed.

Dintenfass [46] reported the case of a man 61 years of age who had chills and presented a positive blood culture (*Streptococcus hemolyticus*) eight days after mastoidectomy. The sinus wall had looked normal. The Tobey-Ayres test showed a partial obliteration of the lumen of the sinus. The patient was treated with daily intravenous injections of 20 cm. of Pregle's iodine, and the temperature gradually returned to normal. Suppuration of the joint of the left elbow developed, which responded to treatment.

In this section it is not amiss to speak of Ricard's [47] observations on traumatic rupture of the lateral sinus. He found that subsequent to injury, chiefly fracture of the skull, a traumatic rupture may occur at the knee of the sinus, and that epidural hemorrhage then develops. The characteristic symptoms are coma, stertorous breathing, progressive hemiplegia, extensive edema of the temporoparietal area, dilated pupil on the injured side and a slow pulse. Trephining over the injured area soon locates the source of the hemorrhage. If it is promptly stopped, the patient may be saved; otherwise he dies of an increased intracranial pressure.

51 West Seventy-Third Street.

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46. Dintenfass, H.: Otitic Pyemia with Jugular Sinus Thrombosis: Recovery Without Ligation, Laryngoscope **39**:731, 1929.

47. Ricard, A.: Des ruptures traumatiques du sinus lateral, Lyon chir. **26**:476, 1929.

Abstracts from Current Literature

Larynx

TANNIN TREATMENT IN CARCINOMA OF THE LARYNX. K. KOFLER, Monatschr. f. Ohrenh. **64**:395 (April) 1930.

Kofler relates that in November, 1919, at a meeting of the Laryngologic Society of Vienna, Weill introduced a patient who had had a tumor on the left vocal cord. Removal of the growth by means of the cold snare was done without difficulty. The hemorrhage was slight and the patient's voice and breathing improved considerably. However, within from two to three months another tumor developed, which again was removed with the cold snare. Histologic examination led to the diagnosis of epithelioma. After removal of the second tumor, Weil began insufflations with a tannin powder. This treatment was given once or twice every week. In the course of the next two years, the patient's condition improved steadily. Examination revealed that the left vocal cord was slightly thicker, otherwise the condition was entirely normal. In another patient with cancer of both vocal cords, Weill likewise gave tannic insufflations, after the largest part of the growth had been removed. In this instance the tannin treatments were given every other day. As a result, the remnants of the growths decreased and within several weeks disappeared entirely. Now only a small scar is visible. The first patient is still well after five years. The second patient recovered more than two years ago, and he has had no relapse. Although most authorities were at first skeptic about Weil's tannin treatment, the author employed it in two instances of squamous cell epithelioma of the vocal cords. He insufflates the tannin powder following anesthetization of the larynx and of the trachea by means of a cocaine-epinephrine solution. In both cases, the tannin treatment was successful. In the conclusion, the author states that observations on four cases do not permit a final decision regarding the effectiveness of the tannin treatment, however, he thinks that it deserves further investigation.

EDITOR'S ABSTRACT.

Nose

PATHOLOGY AND INTRAMURAL ELECTROCOAGULATION OF THE INFERIOR TURBINATE. JOSEPH C. BECK, Ann. Otol., Rhin. & Laryng. **39**:349 (June) 1930.

Since there is a general agreement as to conservatism in the management of the inferior turbinate discussions on this structure are infrequent. The inferior turbinate is a vitally important physiologic structure and must be conserved whenever possible, but its constant enlargement, due to chronic and permanent pathologic changes, is capable of producing symptoms that are annoying to the patient and that require correction. The author classifies the pathologic changes into five types: (1) vascular type, known as turgesence, easily contracted by cocaine or epinephrine, (2) hypertrophy of both the bone and soft parts, not easily contracted by cocaine and epinephrine; (3) the pale, smooth and noncontractile body, so-called hyperplastic or allergic form, usually associated with a similar pathologic process of the middle turbinate and sinuses; (4) atrophic inferior turbinate; (5) neoplastic and other rarer forms of inflammation, as tuberculosis, syphilis and rhinoscleroma.

The principle of intramural electrocoagulation is not new, the object being to secure a decrease in the volume of the soft tissues by the incorporation in them of a shrinking scar. The author believes that this method has many advantages over that of linear cauterization with the electric cautery. The reaction following electrocoagulation is mild and accompanied by little or no discomfiture. Since the

epithelium is not destroyed, no scab or synechia formation is noted. Also, since the coagulating current is used, the vascular beds are sealed, and hemorrhage does not occur.

The technic is carefully described in detail. A standard diathermy machine is used as the source of the current. The turbinal electrode consists of a long Hagedorn needle that has been covered, except for 2 mm. at the tip, by a special dielectric compound. The field being well anesthetized, the needle is plunged into the head of the turbinal in about the center and carried along the medial aspect to the posterior part of the inferior turbinated body, hugging the periosteum. The current is now turned on and the needle slowly withdrawn, the current acting all the while. The same procedure is carried out along the inferior aspect of the turbinate.

ALLERGY AND ITS RELATION TO THE INFLAMMATORY DISEASES OF THE PARANASAL SINUSES. FRENCH K. HANSEL, Ann. Otol., Rhin. & Laryng. **39**:510 (June) 1930.

A careful study of allergy and its relationship to diseases of the paranasal sinus has been made by the author, who concludes that an understanding of this relationship entails the clinical and histopathologic study of vasomotor or hyperesthetic rhinitis, hay-fever, bronchial asthma, catarrhal rhinitis and sinusitis, nasal and paranasal polypi and edema and hyperplastic ethmoiditis. In considering the progress of allergic disease of the nose and sinuses from its onset to the advanced stages of pathologic change, it may be noted that the various terms previously enumerated merely represent different stages of the same disease. Vasomotor or hyperesthetic rhinitis may be considered the early stage of the condition, while the advanced stage may be referred to as hyperplastic sinusitis. The occurrence of acute infection or the establishment of chronic secondary infection frequently complicates the diagnostic problem. Among the factors to be considered in establishing a diagnosis of allergic disease are nasal symptoms, rhinoscopic examination, examination of nasal secretions, roentgenographic study of the sinuses and histopathologic examination of nasal and sinus tissues. A study of the allergic state of the patient should be made with particular reference to the occurrence of other manifestations of allergy, a positive family history of allergy, the presence of positive skin tests and blood eosinophilia. The nasal symptoms in allergy are characterized by sneezing, nasal obstruction and nasal discharge. The discharge varies from thin, serous liquid to thick tenacious mucus, which usually contains eosinophils. The mucous membrane of the nose presents a discolored grayish-pink or bluish-gray appearance, with swelling or edema. In cases with more advanced pathologic process, hyperplasia and polypoid formations may be observed. The histopathologic examination of the nasal and sinus tissues reveals changes similar to those found in the bronchial tissues in bronchial asthma, the surface epithelium being thickened and consisting of many layers of stratified columnar cells. Varying degrees of opacity of the sinuses is revealed by roentgenographic study, the degree of opacity depending on the amount of pathologic change and the presence or absence of retained secretions. Edema and polypi may be shown by the use of iodized poppy seed oil 40 per cent. The presence of transitory edema must be kept in mind, which gives rise to positive changes at one time and negative changes at another.

It may be readily seen that allergic disease with its pathologic changes in the nose and sinuses predisposes to the presence of infection in these structures. The edematous mucosa, with the loss of cilia, causes the retention of secretions and bacteria, which, with added attacks of acute infection, causes a definite suppurative process which may result in chronic infection. The author firmly believes that allergic disease of the sinuses should be accepted as a definite clinical entity and should not be considered in the same light as primary suppurative disease. Although the occurrence of secondary infection in allergic cases places them in a class with the suppurative diseases, even in this instance the primary occurrence of the

allergic process so greatly influences the prognosis and treatment to such an extent that the two groups of cases should be considered separately if satisfactory results are to be obtained. Chronic empyemas may follow in the wake of an allergic process in which an edema of the mucosa has occurred. In the nonsuppurative hyperplastic or catarrhal group of sinus diseases, clinical and histopathologic studies have proved that in the great majority of cases the process is primarily an allergic disease. The author therefore concludes that all hyperplastic, nonsuppurative diseases of the sinus should be considered as of allergic origin until proved otherwise. In the combined suppurative-hyperplastic type, the allergic must be distinguished from the nonallergic. Polypi occur in both types, being more frequent and abundant in allergy than in primary infection. Consequently, they may be recognized as occurring primarily, in one instance, as the result of allergy and in the other as the result of infection. The author believes that many of the disappointments in the treatment of sinus disease have been the result of the failure to make the distinction between allergy, with or without secondary infection, and the true infections or primary suppurations.

SNAPP, Grand Rapids, Mich.

OSTEOMYELITIC PROCESSES IN THE ACCESSORY NASAL CAVITIES. L. HAYMANN, Monatschr. f. Ohrenh. **64**:367 (April) 1930.

In the first part of the article Haymann discusses the terms periostitis, osteomyelitis and osteitis. He points out that there is no essential difference between osteitis and osteomyelitis. These two terms denote only a difference in the localization of the processes. Osteitis may be considered as a special form of osteomyelitis. The author recommends that the term osteomyelitis should not be used in a too narrow sense. It should be applied not only to those processes that are complicated by septic manifestations but also to those cases in which sepsis is absent, but in which the process is extended and has a progressive tendency. The latter conditions exist in nearly all of the eight cases reported by the author. The clinical histories show that the majority of osteomyelitic processes in the maxillary sinus are of dental origin. This proves the necessity of careful aspsis in the treatment of the teeth. However, an inflammation of the accessory nasal cavities may lead to an osteomyelitic process in the wall of the sinus. In a review of the various therapeutic methods, the author expresses the opinion that in cases in which the process is not too progressive incision of the abscesses and light baths of the head, repeated several times a day, often bring favorable results. In most instances, however, an early operation is advisable. Care should be taken that the healthy bone is not infected with the instruments that have come in contact with the diseased area. Occasionally, when the process is limited, an awaiting attitude is permissible, but the opportune time for the operation should not be missed.

PATHOLOGY OF THE NASOPALATINE CANAL. A. SCHOENLANK, Monatschr. f. Ohrenh. **64**:377 (April) 1930.

Schoenlank first discusses the embryology and the anatomy of the nasopalatine canal. He then describes the case of a man, aged 23, in whom he detected an anomaly in the incisive papilla, namely, an entirely open ductus incisivus. By plastic surgery with mucous membrane, the open canal was closed. The author further points out that all types of tumors and of cysts may form in the nasopalatine canal. However, he does not diccuss them in detail. He mentions the fact that by pressing either with the tongue or with the finger on the papilla palatina sneezing may be prevented. This phenomenon may be explained by the innervation. The author further discusses inflammatory disorders of the nasopalatine canal, especially the inflammation of the incisive papilla. He describes two forms. In the first, only the papilla is affected. In the second form the papilla as well as the adjacent portions of the gums are softened, and on pressure,

a serous, saline or a seropurulent fluid is discharged. These inflammations are of gingival, dental or nasal origin. The author also noted an extremely painful condition of the papilla with seropurulent secretion after catarrh or influenza. Gums, palatine mucous membrane and teeth were not involved. For the treatment of these secretory forms, the author recommends irrigations, caustics and light therapy. Massage of the papilla may also be helpful. In cases of dental origin, care of the teeth is necessary.

DIPLOPIA AFTER EXTERNAL OPERATION OF FRONTAL SINUS. ITS PREVENTION AND CURE. M. HALLE, Monatschr. f. Ohrenh. **64**:381 (April) 1930.

Halle points out that since the intranasal operation is employed in the majority of cases of suppurations of the frontal sinus, the external operation has become rare. However, in some instances the external operation is nevertheless necessary, and diplopia frequently results. It has been proposed to avoid this by preserving the trochlea in its original location. But if a radical operation is necessary to avoid relapses, the detachment of the trochlea cannot be avoided, and other methods have to be found to avoid diplopia. The author further describes a surgical method that he employed in correcting diplopia. In the course of recent years, the author performed this operation on twenty patients. The results were perfect in seventeen cases, and in two cases improvement could be noted. Only one operation failed.

EDITOR'S ABSTRACTS.

Miscellaneous

THE DIAGNOSTIC PROBLEM IN ORBITAL CELLULITIS. JAMES A. BABBITT, Ann. Otol., Rhin. & Laryng. **39**:444 (June) 1930.

The differential diagnosis between orbital cellulitis and cavernous sinus thrombosis is extremely difficult and in some instances impossible. It is the field of both otolaryngologist and ophthalmologist, and even their combined efforts may fail. The author concludes that orbital cellulitis is a secondary manifestation of infection and is a symptom-complex rather than an entity. Recent observations reveal that it is almost always due to an infection in the nasal accessory sinuses which has extended into the orbit through venous or lymphatic channels, a dehiscence in the wall, or by means of osseous necrotic changes. The symptoms, together with their character of onset, in both orbital cellulitis and cavernous sinus thrombosis, are described in detail by the author. In comparing the two, it will be noted that exophthalmos and extension to the other eye occur in both. The ptosis of paralysis simulates that of inflammatory edema, while the rigid position of the eyeball due to paralysis of the third, fourth and sixth nerves is practically the same as the fixation due to engorgement of adjacent tissues. Intraocular changes, pain and its reference, failure in vision, symptoms of toxemia or profound sepsis are relatively alike. Laboratory blood studies may be of much help as a very high leukocyte count and a positive blood stream infection would point to the presence of a cavernous sinus thrombosis. The author presents four case reports in which a tentative diagnosis of cavernous sinus thrombosis was made. The accessory sinuses were suspected as a source, and surgical measures were instituted in three cases with the result that definite suppuration was found together with extention into the orbit, most frequently from the ethmoid or frontal sinuses. A complete recovery was encountered in these three cases. Permission for operation was refused in the fourth cases, and death resulted. Autopsy revealed a thick subarachnoid collection of pus over the frontal and frontotemporal region. There was no sign of cavernous or lateral sinus thrombosis. The author concludes that the serious prognosis without intervention justifies initial operation on the nasal sinus, even in cases of doubt.

PALATAL PALPEBRAL REFLEX. CHARLES J. IMPERATORI, Ann. Otol., Rhin. & Laryng. **39**:458 (June) 1930.

A reflex for determining the tonus of the musculature of the soft palate has been observed and described by the author. It is one that is observed as a normal reaction and consists of a blinking of the upper eyelids on stimulation of the soft palate. Its absence, excepting in rare instances, may be considered as abnormal. The reaction is induced by a percussion of the soft palate with a small stick applicator, the point of the stick being applied midway between the midportion of the uvula and the junction of the palate with the anterior tonsillar pillar. The reaction is bilateral and is only rarely noted on the side of percussion. No vomiting reflex has been induced in any instance. No result is obtained on stimulation of the inner side of the cheek, tongue, hard palate or base of the tongue. Several explanations have been advanced as to the exact mechanism involved in the reflex, the exact nature of which has not yet been determined. Apparently there are two elements present: a deep or muscular one and secondarily, a sensory one. From the cases examined it would seem that a deep or muscular element was the more important one. The value of the procedure is (1) to determine the tonus of the musculature of the soft palate; (2) to detect slight paralysis of the soft palate and (3) to differentiate between a sensory and muscular paralysis in this region.

SNAPP, Grand Rapids, Mich.

DENKER'S OPERATION IN TREATMENT OF CYSTS OF THE MAXILLA. T. ZALEWSKI, Monatschr. f. Ohrenh. **64**:386 (April) 1930.

Zalewski states that smaller cysts of the upper teeth are usually treated by the stomatologist whereas larger cysts are treated by the rhinologist. Numerous operative methods have been employed. They are of two types. Those of the first type establish a communication of the cyst with the mouth, those of the second type with the nasal cavity. Most specialists operate according to the methods of the second group. The author further describes and evaluates several methods, namely, Partsch's, Caldwell-Luc's and Denker's. He, himself, employed Denker's method in five cases. The cyst cavity is opened with the typical incision. After that, a connection is established between the cyst cavity and the nose, without removing the cyst. Following disinfection of the cyst cavity, a tampon is inserted. After two days the tampon is removed, and the cavity is irrigated daily until it remains dry. All five patients recovered quickly. The treatment required from fourteen to twenty-six days. Denker's operation is advisable especially in cases in which the cyst is on the outer wall of the nasal cavity.

THERAPEUTIC EFFECTS OF SMALL DOSES OF IODINE IN SECRETORY DISORDERS (HYPERSECRETION, HYPOSECRETION, VASOMOTOR RHINITIS) OF THE MUCOUS MEMBRANE OF THE RESPIRATORY PASSAGES. H. STERNBERG, Monatschr. f. Ohrenh. **64**:401 (April) 1930.

Sternberg shows how iodine influences the mucous membrane of the respiratory passages, what therapeutic results may be obtained and the significance for diagnosis and for biologic observations. In very small doses (hundredth parts of a milligram), iodine usually reduces the tissue permeability of the mucous membrane; it reestablishes normal permeability. The larger the dose of iodine, however, the greater is the tendency that the tissue permeability is increased (iodine coryza, iodine acne). Larger amounts of iodine are administered for stimulation of the secretion and to make the secretion more aqueous. The threshold dose of iodine at which this change in the modification of the tissue permeability takes place is undetermined and differs in various patients. At any rate, already a comparatively small quantity of iodine may increase the tissue permeability. The author thinks that the favorable influence of iodine on sec-

ondary inflammatory swellings near specific processes, such as cancers, tuberculosis of the respiratory tract and gummas, is probably due to a reduction, regulation or stimulation of the tissue permeability, which is always abnormal in inflammatory processes. A specific action of iodine on the pathogenic microorganisms is not probable. In the conclusion, the author states that our knowledge on the influence of iodine on the mucous membrane of the respiratory passage is still incomplete and that further investigations will be necessary.

HISTOLOGIC EXAMINATION OF FIFTEEN YEAR OLD BONE TRANSPLANT. D. N. MATWEJEW, Monatschr. f. Ohrenh. **64**:417 (April) 1930.

Matwejew reports the case of a patient, aged 35, who at the age of 15 had had lupus of the face. The patient had received treatment, scars had formed and relapses did not develop. Several years later, in order to repair the defect of the nose, a piece from the tibia was transplanted under the skin of the left shoulder according to the Italian method of rhinoplasty. The author describes the present status of the patient and the results of the microscopic examination of the transplant. It was found that the bone tissues had not simply grown into the new location but had first undergone changes. This observation is in accordance with the opinion of most authorities. The new bone is different from the original transplant. The periosteum, the endosteum, the bone-marrow and perhaps also the surrounding tissues serve as regenerative elements.

EDITOR'S ABSTRACTS.

Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

April 7, 1930

SAMUEL SALINGER, M.D., *President, in the Chair*

RHINOPHYMA. DR. SAMUEL SALINGER.

Last fall I presented before this society, a case of an unusually large rhinophyma. I thought at the time that it was the largest I would ever see. Today I present a similar case in which the growth is even larger than the previous one (fig. 1).

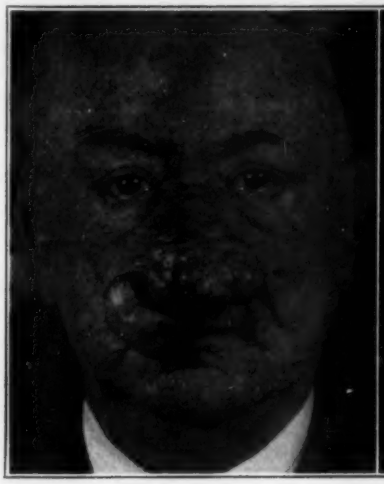

Figure 1

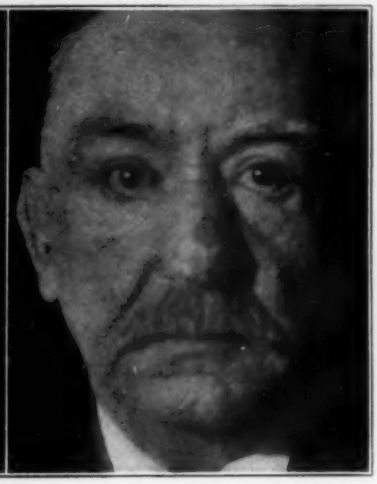

Figure 2

One of the lobules which hung over the lower lip was so firm that I suspected malignant degeneration. Histologic section, however, proved otherwise. The operation was attended with comparatively little bleeding. Subsequently epithelial grafts were employed which helped materially in the cosmetic result (fig. 2).

PRIMARY CARCINOMA OF THE MASTOID WITH CHOLESTEATOMA. DR. T. C. GALLOWAY.

A patient was presented who had primary carcinoma of the mastoid with cholesteatoma.

RETICULO-ENDOTHELIAL SYSTEM AS A DEFENSIVE MECHANISM. DR. PAUL R. CANNON.

The conception of a generalized system of cells of the connective tissues which are characterized by their frequency and intensity of phagocytosis was popularized by Aschoff under the name of the reticulo-endothelial system. The designation of a so-called system is founded on the monumental work of Metchnikoff, later amplified by many workers. With the advent of vital dyes, it became possible to show that colloidal and particulate materials are more readily removed by some cells of the connective tissues than by others, and that these cells are more

abundant in such organs as the spleen, liver, bone-marrow, omentum, lymph nodes, suprarenals, hypophysis and, to a lesser extent, wherever loose connective tissue exists. This distribution may be demonstrated by injecting a suspension of particulate material such as india ink intravenously into an animal and finding the ink soon concentrated in the spleen, liver and bone-marrow, and to a lesser degree in lymphoid and connective tissue. Practically none will be found in muscles, or in tissues where the blood supply is rapid.

Bacteria are particulate particles when in the blood stream and they also are removed principally by the phagocytes or macrophages of the spleen, liver, bone-marrow, etc. In this way the body successfully defends itself against ordinary disseminations of infectious agents. The organisms are ingested and digested by the macrophages and the infection is terminated. All states of immunity are relative, however, and the effectiveness of the defense will depend on the number and virulence of the organisms and probably also on the functional state of the macrophages. At times, the system of macrophages is inadequate to handle the infection and death may occur.

One of the problems of interest in connection with the property of phagocytosis of the macrophages is the possible relationship between it and the formation of immune bodies. Metchnikoff believed that the antibodies were merely products resulting from the digestion of protein substances by phagocytic cells, both microphages and macrophages. Later work has tended to minimize the importance of the microphages but has strengthened the view that the macrophages are the principal if not the sole formers of antibodies.

This belief has arisen because when organs richest in macrophages are removed, such as the spleen, the antibody production is significantly decreased. Also when the hemopoietic tissues are injured by chemical and physical agents, such as benzene, thorium-X and x-rays, the production of antibodies is diminished. Extracts of organs richest in macrophages also show the greatest concentration of antibodies. In tissue culture it has been shown that immune bodies are formed by macrophages. In recent times, experiments aiming to exclude a large number of macrophages from functional activity by means of so-called blockade have shown an inverse relationship between the concentration of antibodies in the blood stream and the degree of blockade of the phagocytic cells. All of these facts supplement one another and indicate that the macrophages are potent sources of antibodies.

The importance of these facts is apparent, as it furnishes a means of determining the relative value of cellular and humoral factors in the mechanism of defense. When one compares the transitory state of passive immunization with the permanent degree of active immunity it becomes obvious that in the latter type antibodies in the blood stream must play a small part. And indeed in natural immunity there is little evidence that antibodies are of significant value. On the other hand, evidence indicates that in many infectious diseases the resistance depends primarily on the numbers and functional activity of the macrophages, which lead to a relatively permanent active immunity. This does not exclude the participation of antibodies but suggests that they are of most importance intracellularly rather than in the blood stream.

Within recent years Besredka has written a book on the subject of local immunity in which he attempts to prove that immunity of local tissue may be achieved in the entire absence of antibodies in the blood, and by implication, in the tissues themselves. His theories have been received with interest, but have not been confirmed by experimental facts. Nevertheless, Besredka has demonstrated that there is such a thing as local immunity of tissues, and has applied his ideas to the treatment for local infections of the skin and mucous membranes. His conception is that when bacteria are grown in broth, an antagonistic substance is formed which is specific and gradually stops the growth of the organisms. To this hypothetic substance he has given the name antivirus. By the use of broth dressings and injections of this antivirus in tissues he believed that certain cells absorbed it and thus became immune to the bacteria appearing there later. This antivirus has had a wide clinical use in France and is now on the market in the United States.

Mr. Pacheco and I have been interested in this problem of local immunity and have performed experiments in an attempt to analyze the mechanism. We have found that when a staphylococcus vaccine of killed organisms is injected intradermally for several injections and the animal is then allowed to rest for from three to four weeks, the later injection of living virulent staphylococci into this region is followed by a localized, small abscess with no serious consequences to the host. The injection of a similar quantity of these living virulent staphylococci into the skin of a normal animal is followed by cellulitis and death in most cases. We have, then, a local immunity produced by active immunization. In studying the tissues of such animals we find that whereas the staphylococci diffuse throughout the subcutaneous tissues in the normal animals and are vigorously phagocytized by polymorphonuclear leukocytes, nevertheless the infection generalizes and death occurs. On the other hand, in the immunized animal there has been a marked increase in the number of macrophages in the subcutaneous tissues, and these cells are extremely active in phagocytosis in the reinfection. In addition, there is evidence that in the immunized skin there is agglutination of staphylococci with a consequent fixation of the organisms at the site of inoculation, so that they are then more readily disposed of by the macrophages of that vicinity.

The local immunity seems to be due to two conditions: a local fixation of the bacteria, probably by a specific antibody present locally, and reinforced by the phagocytic activities of increased numbers of macrophages there present. This illustrates again a statement of Opie, that by this process "vital organs are protected at the expense of local injury."

DISCUSSION

Dr. William Bloom: Several important points can be gleaned from Dr. Cannon's presentation. He has shown clearly that if a large number of reticulo-endothelial cells are put out of working order by india ink, few antibodies are liberated into the blood. He has also shown the reaction of these cells on the reinjection of the inflammatory agent, and has reached a definite, objective conclusion. Such work is in sharp contrast to the tremendous amount of speculative work which is written about the "reticulo-endothelial" system so freely, particularly in clinical literature. When the "reticulo-endothelial" system is made responsible for everything that takes place in the body, it is refreshing to find that some objective careful work can be done on these cells. Perhaps the best point made so far is their connection with inflammation, that is, the "local defense reaction." There is some evidence to indicate that these cells are involved in lipoid metabolism, but other than this I think there is nothing to prove definitely that these cells participate in the metabolism of the body. This applies particularly to the part these cells might or might not play in any carcinomatous involvement. The big difficulty as I see it, and as I think Dr. Cannon has found, is that in many of the processes that take place in the body, one is dealing with antigens in solution, which are invisible. Until some way of demonstrating them with the microscope or otherwise is found, it will be hard to link up the allergens with the "reticulo-endothelial" system.

Dr. J. Holinger: What is the relation of these observations to Professor Bier's hyperemia? If one injects from five to ten times a fatal dose of different kinds of poison or even of infectious material into the leg of a dog, after the leg has been tied up with an Esmarch bandage, the isolated leg will overcome the poison or infection and the dog will develop no general symptoms. Has the manner in which this infection is overcome anything to do with the liberation of antibodies?

Dr. Frank Novak: In reference to the illustration that was presented showing an increased number of macrophages, I think that the mechanism of immunity might be explained on the basis of phagocytosis, but I wish to know what connection the immune bodies have with it.

Dr. Alfred Lewy: What evidence exists that the body fluids have to do with the bacteria, perhaps preliminary to their being ingested by the macrophages?

DR. BUDD C. CORBUS: After reading Besredka's book it occurred to me that I might accomplish something with the gonococcus. After great difficulty I have grown the gonococcus bouillon with ascites fluid. My associate and I began our experiments first by injecting the urethra with gonococcal bouillon filtrate and sealing it in, but there was such a tremendous reaction to this that we had to stop the experiments. We then began to give intradermal injections, and were surprised to find that a splendid response occurred with proper dosage in clearing up the urethral discharge. We have treated some fifty or sixty patients before presenting a preliminary report to the Chicago Urological Society.

The first response noticed was an enormous exudate in which many intracellular cocci were found, later becoming extracellular. As the process goes on to extermination, the urine becomes clearer, there are a few threads showing a few polymorphonuclear cells, and finally these disappear and the patient recovers. We have given as high as 2 cc. We have taken the leukocyte count in all cases and found in some instances from 2,000 to 4,000 increase. In every case in which there was a positive complement-fixation test, there was a strong response after the third week of treatment. We tested this out to see how long the antibody response persists.

I attended the meeting because of my great interest in the subject of the reticuloendothelial cells, and I express my gratitude to Dr. Bloom and Professor Cannon for their helpful presentation.

DR. PAUL R. CANNON: In reference to phagocytosis, there is one fundamental feature, which is that probably phagocytosis cannot occur to any extent in circulating fluids. The very nature of the phenomenon demands a degree of quiet. It occurs mainly in organs that have a rather slow blood or lymph flow, as in the spleen, bone-marrow, liver and lymph nodes. Where the current is stagnant and particles come in contact with a phagocytic cell there is an opportunity for them to be taken up. I suspect that the favorable effects of Bier's passive hyperemia in the example cited might be due to the fact that phagocytosis can occur where there is quiet, whereas under normal conditions many of the bacteria would be carried elsewhere. The mechanism of cellular immunity is also of importance in tuberculosis. The conceptions of the defense in tuberculosis have been disturbed in the past year. It has been held that the reason a tuberculous reinfection remains localized is that there is an infiltration of cells which exert a barrier-action and seal off the tubercle bacilli. Recent work has tended to indicate that this explanation may not be true. An important principle has been developed by Opie in the demonstration of the mechanism of anaphylactic inflammation as a reaction to the meeting of antigen and antibody in the tissues. According to this explanation, the cellular infiltration is secondary rather than primary, as is usually believed. In the work on skin immunization just described there is definite evidence in the immune animals of an actual agglutination of the organisms in vivo, whereas in normal animals no such reaction occurs. I believe that antibodies play an important part in the process. It is known that they are not the sole element, however.

One interpretation is that phagocytic cells, once having had experience with a particular antigen acquire an ability to digest it, so that later it may be digested with more rapidity. It is possible that this is due to the fact that tissues develop an ability to form antibodies more rapidly in later immunizations. There is much objective evidence supporting this view.

As to how the antibodies may act, the current conception of antibodies in general is tending toward the view that many antibodies are of the same type, working in different ways, the agglutinating factor, the precipitating factor and the opsonizing factor all being different manifestations of one general force.

In regard to the work on the gonococcus, it is known that with most organisms, the body can be specifically sensitized so that when the organisms are reinjected more reaction occurs than on the first occasion. It seems to me a logical treatment to use specific preparations in attempting to stimulate cells that are working against the gonococcus and are keeping it localized. By the specific irritation which is produced, perhaps more of the cells may be mobilized and brought to the

point of infection, and thus aid in phagocytosing the organisms. I believe there are logical grounds for using such a treatment as Dr. Corbus has mentioned.

Nasal Allergy as Related to Hyperesthetic Rhinitis and Hay-Fever.

Dr. Samuel Feinberg.

Hyperesthetic rhinitis and hay-fever should be placed in the category of important disease because of the frequency of their occurrence, the annoyance of their symptoms, the disability which they cause and the common occurrence of complications, such as sinus disease and asthma. There can be little doubt that the allergic concept explains satisfactorily a large proportion of the instances of hyperesthetic rhinitis. I do not contend that this is the only possible theory nor that this concept explains all phases or all cases. The allergic concept presupposes a constitutional basis which allows particular persons to become sensitized; of this basic constitutionality little is known. The immediate exciting agents in hyperesthetic rhinitis are the pollens, animal epidermal substances, foods, miscellaneous materials and possibly bacteria. Secondary etiologic factors are mechanical (dusts, polypi), thermal, neurogenic and infectious.

The diagnosis of hyperesthetic rhinitis can be complete only if the tests for the specific exciting causes are made. This is usually done by cutaneous tests, but at times also intracutaneous, nasal or conjunctival tests are indicated. There are many pitfalls in the interpretation of these tests and they should be considered only in conjunction with clinical and therapeutic trials. Positive tests are obtained in about 99 per cent of seasonal cases of hay-fever and 30 to 50 per cent of perennial hyperesthetic rhinitis.

In the majority of cases, with proper attention to certain principles, the treatment of the patient with seasonal hay-fever and the allergic type of hyperesthetic rhinitis is successful. Treatment in the allergic cases aims at the elimination of the offending substances when feasible and desensitization when the former is not possible. Most of the failures are in the group of nonallergic cases. Here vaccine and local therapy have been the methods chiefly used. It is proposed that what we are now designating as hyperesthetic rhinitis may represent more than one clinical entity. It is possible that by closer cooperation between allergists and rhinologists, greater progress could be made in this important field.

DISCUSSION

Dr. Frank Novak: I am familiar with Dr. Feinberg's work, and I stress two points; first, the necessity of drawing a sharp line between the two conditions of hyperesthetic rhinitis and hay-fever. They should not be used interchangeably because they are two different conditions. Some cases of hyperesthetic rhinitis are allergic, but the majority are nonallergic. They have an endocrine basis.

Desensitizing procedures should be carried out by a trained person who knows the subject of allergy, and not by one who has received his training from a representative of a pharmaceutic house, for the work is not innocuous. Some fatalities have been reported, even in the hands of experienced persons. I believe that the specialty should be practiced only by those who devote their time entirely to such work, and that others should refer their patients to such persons for testing.

Dr. Leon Unger: I worked with Dr. Feinberg for many years in this field and can corroborate his statements. I am sure that Dr. Feinberg treated the subject in a conservative manner and was not over-enthusiastic, as many specialists are. I believe that his figures in the results obtained in hyperesthetic rhinitis were accurate. We have had a considerable percentage of good results and a considerable number of failures in the treatment of nasal allergy, but the longer we work the better are the results. We are becoming more and more convinced that skin tests must be corroborated by other methods of testing and that one cannot say a patient is not allergic because the skin tests show negative results. A small amount of some irritant may have no effect on a patient, whereas if a larger amount is used, his threshold may be overstepped. The threshold idea, I think, also explains

the better results obtained when complete skin tests are made. Those specializing in sensitization work should work more in harmony with otolaryngologists.

DR. T. C. GALLOWAY: Many of the infectious conditions in the nose, especially sinusitis in children, are superimposed on an allergic state. I am surprised that Dr. Feinberg gave such a low percentage of positive results. Amazing results have been obtained when the possibility of an allergy in patients seen for obstinate infection has been considered. A typical case was seen in which there was first otitis media and then disabling infections every few weeks over a long period, with the mucous membranes red and inflamed every time. Finally in a clear interval the characteristic pale, boggy membranes of allergy were noted, and, although two sets of skin tests gave negative results, trial determined a sensitiveness to egg. When the allergic factor was removed, the child's infections stopped completely.

I think that the profession will hear more and more of allergy all the time, and that the society is fortunate in having such a scientific paper presented.

DR. WALTER H. THEOBALD: I am glad that Dr. Feinberg has brought up the neurogenic type of case. In many such patients under my observation skin tests are negative, but symptoms of hyperesthetic rhinitis occur any time of the year. In my experience these cases are greatly improved and many times cured by cauterization of the so-called Killian sensitive points. Dr. Norval H. Pierce has reported many such cases in which there have been no allergic observations.

DR. SAMUEL FEINBERG: Many patients with hay-fever also have a hyperesthetic rhinitis lasting throughout the year. Physicians specializing in this work see many patients who come to them with a diagnosis of hyperesthetic rhinitis and discover marked positive pollen tests. Then the patient often recalls that his symptoms are more pronounced at certain seasons of the year, particularly those associated with pollens.

I am impressed with the idea that there is some association of asthma with hyperesthetic rhinitis, and of hay-fever with hyperesthetic rhinitis. I am glad that the point concerning positive skin tests was appreciated, for if one depends on skin tests alone for a definite diagnosis, many failures will result. Many tests should be carried out and the results correlated with the clinical observations and trials. I think that Dr. Galloway misunderstood me in regard to the percentages cited, as I referred only to the hay-fever group.

Directory of Otolaryngologic and Ophthalmologic Societies *

NATIONAL

American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Dr. T. E. Carmody, 227, 16th St., Denver.
Secretary: Dr. Gabriel Tucker, Bronchoscopic Clinic, University Hospital, Philadelphia.
Place: Philadelphia. Time: 1931.

American Academy of Ophthalmology and Oto-Laryngology

President: Dr. William H. Wilder, 122 S. Michigan Ave., Chicago.
President-Elect: Dr. J. F. Barnhill, Miami Beach, Fla.
Executive Secretary: Dr. William P. Wherry, Medical Arts Bldg., Omaha.
Place: Chicago. Time: Oct. 27-31, 1930.

American Bronchoscopic Society

President: Dr. Henry B. Orton, 24 Commerce St., Newark, N. J.
Secretary-Treasurer: Dr. Louis H. Clerf, 128 S. 10th St., Philadelphia.

American Laryngological Association

President: Dr. Francis R. Packard, 304 S. 19th St., Philadelphia.
Secretary: Dr. George M. Coates, 1721 Pine St., Philadelphia.

American Laryngological, Rhinological and Otological Society

President: Dr. Max A. Goldstein, 3858 Westminster Pl., St. Louis.
Secretary: Dr. R. L. Loughran, 33 E. 63d St., New York.

American Otological Society

President: Dr. David H. Walker, 101 Bay State Rd., Boston.
Secretary: Dr. Thomas J. Harris, 104 E. 40th St., New York.

SECTIONAL

Academy of Medicine of Northern New Jersey, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Frederick J. Wort, 1080 Broad St., Newark.
Secretary: Dr. Earl LeRoy Wood, 31 Lincoln Park, Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. G. G. Marshall, 122 West St., Rutland, Vt.
Secretary-Treasurer: Dr. Arthur F. Holding, 142 Washington Ave., Albany.
Time: Third Wednesday of October, November, March, April, May and June.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

NEW ENGLAND OTOLOGICAL AND LARYNGOLOGICAL SOCIETY

President: Dr. Owen Smith, 690 Congress St., Portland, Maine.
Secretary-Treasurer: Dr. Stanwood Fisher, 192 State St., Portland, Maine.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. Arthur C. Jones, 105 N. 8th St., Boise, Idaho.
Secretary-Treasurer: Dr. Walter F. Hoffman, 817 Summit Ave., Seattle.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Walter F. Hoffman, 817 Summit Ave., Seattle.
Secretary-Treasurer: Dr. Francis A. Brugman, Medical-Dental Bldg., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Fred Hasty, 119 Seventh Ave., Nashville, Tenn.
Secretary: Dr. Fletcher D. Woodward, Box 162, University, Va.
Place: Louisville, Ky. Time: November, 1930.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. Wilfrid Haughey, 65 W. Michigan Ave., Battle Creek.
Secretary: Dr. R. H. Fraser, Battle Creek Sanitarium, Battle Creek.
Time: Third Thursday of alternate months.

STATE

COLORADO OTO-LARYNGOLOGICAL SOCIETY

President: Dr. C. E. Cooper, 227, 16th St., Denver.
Secretary: Dr. Rex L. Murphy, Metropolitan Bldg., Denver.
Place: Assembly Room of Metropolitan Bldg. Time: First Saturday of each month from October to May.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Dorland Smith, 836 Myrtle Ave., Bridgeport.
Secretary-Treasurer: Dr. Walter L. Hogan, 750 Main St., Hartford.

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President: Dr. George B. Smith, Rome.
Secretary-Treasurer: Dr. William O. Martin, Jr., 384 Peachtree St., Atlanta.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. H. A. Van Osdol, 320 N. Meridian St., Indianapolis.
Secretary: Dr. Robert J. Masters, 23 E. Ohio St., Indianapolis.
Place: Indianapolis. Time: Dec. 10 and 11, 1930.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. C. N. Spratt, 900 Nicollet Ave., Minneapolis.
Secretary-Treasurer: Dr. J. H. Morse, 80 S. 7th St., Minneapolis.

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY

President: Dr. Lee W. Smith, 125 W. Granite St., Butte.
Secretary: Dr. J. G. Parsons, Lewistown.
Time: Semi-annually.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. H. B. Beeson, 232 W. De Mers Ave., Grand Forks.
Secretary-Treasurer: Dr. F. L. Wicks, Valley City.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. J. W. McCollom, 346½ Washington St., Portland, Ore.
Secretary: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland, Ore.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. J. E. Raia, 272 Broadway, Providence.
Secretary: Dr. F. W. Dimmitt, Jr., 195 Thayer St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. F. M. McHugh, 17 Exchange Pl., Salt Lake City.
Secretary-Treasurer: Dr. W. Leroy Smith, Ezra Thompson Bldg., Salt Lake City.

VIRGINIA SOCIETY OF OTOLARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Fletcher D. Woodward, Box 162, University.
Secretary-Treasurer: Dr. Henry Grant Preston, Harrisonburg.

LOCAL

ATLANTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. William L. McDougall, Atlanta National Bank Bldg., Atlanta.
Secretary: Dr. William O. Martin, Jr., 384 Peachtree St., Atlanta.
Place: Academy of Medicine, 38 Prescott St. N. E. Time: 8 p. m., last Thursday of each month.

BALTIMORE CITY MEDICAL SOCIETY, SECTION ON OTOLOGY AND LARYNGOLOGY

Chairman: Dr. Waitman F. Zinn, Medical Arts Bldg., Baltimore.
Secretary: Dr. Moses L. Breitstein, 1212 Eutaw Pl., Baltimore.
Place: 1211 Cathedral St. Time: 8:30 p. m., last Friday of each month.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Samuel Salinger, 25 E. Washington St., Chicago.
Secretary-Treasurer: Dr. Austin A. Hayden, 25 E. Washington St., Chicago.
Place: Medical and Dental Arts Bldg., 185 N. Wabash Ave. Time: 6 p. m., first Monday of each month from October to May.

CINCINNATI OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Edward D. King, 707 Race St., Cincinnati.
President-Elect: Dr. M. F. McCarthy, Union Central Bldg., Cincinnati.
Secretary-Treasurer: Dr. Samuel Seltz, Provident Bank Bldg., Cincinnati.
Place: University Club. Time: 6:45 p. m., second Tuesday of each month from October to May.

Cleveland Academy of Medicine, Section of Ophthalmology and Otolaryngology

Chairman: Dr. M. Paul Motto, Rose Bldg., Cleveland.
Secretary: Dr. A. D. Ruedemans, Euclid and E. 93d St., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

College of Physicians of Philadelphia, Section on Otolaryngology

Chairman: Dr. George M. Coates, 1721 Pine St., Philadelphia.
Clerk: Dr. Frederic M. Strouse, 1301 Spruce St., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., third Wednesday of each month from October to June.

Columbus Ophthalmological and Otolaryngological Society

Chairman: Dr. John E. Brown, 370 E. Town St., Columbus, Ohio.
Secretary-Treausurer: Dr. Albert D. Frost, 681 E. Broad St., Columbus, Ohio.
Time: First Monday of each month.

Dallas Academy of Ophthalmology and Otolaryngology

President: Dr. H. B. Decherd, 1717 Pacific Ave., Dallas, Texas.
Secretary: Dr. W. Mood Knowles, 1717 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Detroit Oto-Laryngological Society

President: Dr. C. C. McClelland, David Whitney Bldg., Detroit.
Secretary-Treasurer: Dr. Don A. Cohoe, 13535 Woodward Ave., Detroit.
Place: Maccabees Bldg. Time: Third Wednesday evening of each month from October to May.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. W. R. Thompson, 510 Main St., Fort Worth, Texas.
Secretary-Treasurer: Dr. R. H. Needham, Fort Worth National Bank Bldg., Fort Worth, Texas.
Place: University Club. Time: 6:30 p. m., first Friday of each month except July and August.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. Claude C. Cody, 1306 Walker Ave., Houston, Texas.
Secretary: Dr. J. M. Robinson, Eye, Ear, Nose and Throat Hospital, Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., first Thursday of each month from September to June.

Indianapolis Ophthalmological and Oto-Laryngological Society

President: Dr. E. L. Lingeman, 241 N. Pennsylvania St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, Hume-Mansur Bldg., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Otolaryngology

President: Dr. A. E. Eubank, Arygle Bldg., Kansas City, Mo.
Secretary: Dr. O. S. Gilliland, Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

President: Dr. Dean E. Godwin, Security Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. Ferris Arnold, Security Bldg., Long Beach, Calif.
Place: Pacific Coast Club. Time: Last Wednesday of each month from September to June.

Los Angeles County Medical Society, Section of Ophthalmology and Otolaryngology

President: Dr. A. Ray Irvine, 727 W. 7th St., Los Angeles.
Secretary-Treasurer: Dr. J. Frank Friesen, 727 W. 7th St., Los Angeles.
Place: California Hospital, assembly room. Time: 8 p. m., first Monday of each month.

Medical Society of the District of Columbia, Section on Ophthalmology and Oto-Laryngology

Chairman: Dr. James A. Flynn, 1511 Rhode Island Ave., Washington.
Secretary: Dr. E. R. Gookin, Medical Science Bldg., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month.

Memphis Society of Ophthalmology and Oto-Laryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

The Milwaukee Oto-Ophthalmic Society

President: Edward R. Ryan, 425 E. Water St., Milwaukee.
Secretary-Treasurer: Dr. O. P. Schoofs, 120 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. R. J. Warner, 706 Church St., Nashville.
Secretary-Treasurer: Dr. W. W. Wilkerson, Jr., Bennie-Dillon Bldg., Nashville.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month.

New Orleans Ophthalmological and Otological Society

President: Dr. William A. Wagner, Pere Marquette Bldg., New Orleans.
Secretary-Treasurer: Dr. M. F. Meyer, Canal Bank Bldg., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: January to June and October to January—third Thursday of each month.

New York Academy of Medicine, Section of Laryngology and Rhinology

Chairman: Dr. John M. Loré, 240 West End Ave., New York.
Secretary: Dr. Robert E. Buckley, 2 E. 103d St., New York.
Place: 103d St., and 5th Ave. Time: 8:30 p. m., fourth Wednesday of each month from October to May.

New York Academy of Medicine, Section of Otology

Chairman: Dr. Clarence H. Smith, 2 E. 54th St., New York.
Secretary: Dr. James Garfield Dwyer, 57 W. 57th St., New York.
Place: 103d St. and 5th Ave. Time: Second Friday of each month from October to May.

Omaha and Council Bluffs Ophthalmological and Otolaryngological Society

President: Dr. Charles M. Swab, Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. J. C. Davis, 1615 Howard St., Omaha.
Place: Medical Arts Tea Room. Time: 5:15 and 7 p. m., third Wednesday of each month from October to May.

Philadelphia Laryngological Society

President: Dr. Arthur J. Wagers, 1429 Spruce St., Philadelphia.
Secretary: Dr. Herman B. Cohen, 1301 Spruce St., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., first Tuesday of each month from October to June.

Pittsburgh Otological Society

Chairman: Dr. Watson Marshall, 121 University Pl., Pittsburgh.
Secretary-Treasurer: Dr. Thomas B. McCollough, 121 University Pl., Pittsburgh.

Rochester Eye, Ear, Nose and Throat Society

Chairman: Dr. A. Vincent Walker, 833 South Ave., Rochester, N. Y.
Secretary-Treasurer: Dr. John F. Gipner, 53 Fitzhugh St., Rochester, N. Y.
Place: Rochester Medical Association, 13 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ear, Nose and Throat Club

Acting President: Senior member of the executive committee: Dr. George E. Hourn, 607 N. Grand Blvd., St. Louis.
Secretary: Dr. Arthur M. Alden, Frisco Bldg., St. Louis.
Place: University Club Bldg. Time: Third Wednesday in February, April, October and December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. F. W. Sorrell, Gibbs Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. A. F. Clark, 728 Main Street, San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

Spokane Academy of Ophthalmology and Otolaryngology

President: Dr. A. N. Codd, 421 W. Riverside Ave., Spokane, Wash.
Secretary: Dr. Marc Anthony, Old National Bank Bldg., Spokane, Wash.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. Searle B. Marlow, State Tower Bldg., Syracuse, N. Y.
Secretary-Treasurer: Dr. A. Harry Rubenstein, Medical Arts Bldg., Syracuse, N. Y.
Place: University Club. Time: Second Friday of each month except June, July and August.